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ORALISM AND AURALISM

A QUARTERLY JOURNAL DEVOTED EXCLUSIVELY TO PROBLEMS OF THE

DEAF AND DEFECTIVE SPEECH

EDITOR

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St. Louis

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THE
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Vol. XXXIII ST. LOUIS, MAY, 1923 No. 5

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

DETERMINATION OF THE LINE OF THE DESCENDING PORTION OF THE FACIAL CANAL IN DOING THE MASTOID OPERATION.*

(Preliminary Report in order to Establish Priority.)

DR. FRANK BRIDGETT, Philadelphia.

While making a series of specimens of the macerated temporal bone, I made some observations pertaining to the determination of the position of the descending portion of the facial nerve.

In doing the mastoid operation, either the simple or radical, knowing that the earlier operators laid down certain rules to be followed, having mapped out certain areas, and having picked out anatomical landmarks that would serve as guides, the operator can accordingly proceed with a degree of safety.

Macewen in his work ("Diseases of the Brain and Spinal Cord") states: "The mastoid antrum is the key to the position in all operations whose aim is to expose the mastoid cells. Once the antrum has been opened, the further enlargement of the aperture in the direction of the mastoid cells may be undertaken with safety, and through the passage between the antrum and middle ear or by its enlargement, the latter may be thoroughly cleansed.

The main point in the operation is to expose the antrum, and while doing so to avoid—first, opening the sigmoid groove and injuring its enclosed sinus; secondly, encroaching upon the Fallopian canal, and destroying the facial nerve; thirdly, invading the

*Read before the Philadelphia Laryngological Society, April 18, 1922.
*Accepted for publication in "The Laryngoscope," May 20, 1922.

middle fossa of the skull; and fourthly, injuring the semi-circular canals."

Following these cardinal principles, therefore, in doing the mastoid operation, we have the following anatomical landmarks that must be recognized in order that the result of the operation may be successful.

First, Macewen mapped out the supermeatal triangle on the post-auricular process of the squamous portion of the temporal bone, an area corresponding externally to the location of the antrum tympanicum internally, and the boundaries are as follows: "The upper border formed by the posterior root of the zygoma running somewhat horizontally above, the portion of the descending plate of the

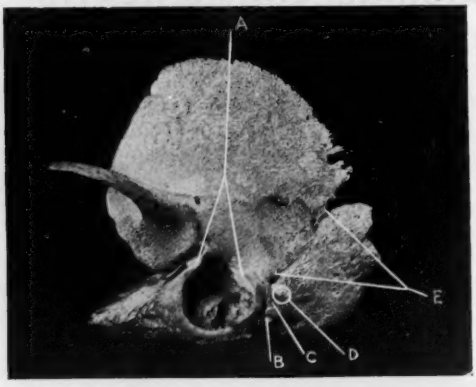


Figure 1. Outer aspect of left temporal bone of the new born. Pictures best studied with binocular loupe. A, Annulus tympanicus. B, Styloid process and stylomastoid foramen immediately behind and a mere suggestion of the digastric groove. C, Future tympanomastoid fissure. D, Mastoid tubercle. E, Petrosquamous suture.

squamous which forms the arch of the osseous part of the external auditory meatus below, and a base line uniting the two, dropped from the former on a level with the posterior border of the external auditory meatus." This triangle, after having been opened, together with the obliquity of the posterior superior osseous wall of the external auditory canal and the depth of the inner wall of the tympanic cavity as determined by passing a probe along the posterior wall of the external auditory canal from the level of the external auditory meatus, the tympanic membrane having been previously perforated by disease, the foregoing landmarks point the way to the successful entrance to the antrum.

When I state that it gives us the location of the antrum I must necessarily say that it also gives us the location of the external semi-circular canal and the beginning of the descending portion of the aqueductus Fallopii which are on the inner wall of the antrum at this point.

Secondly, we have the linea temporalis together with its continuation, the supramastoid crest, seen on the squamous portion of the temporal bone leading back to the notch at the articulation of the squamous with the petromastoid portion of the temporal bone, giving us a line externally which approximately corresponds to the tegmen tympanii and tegmen antri internally, a plate of bone serv-

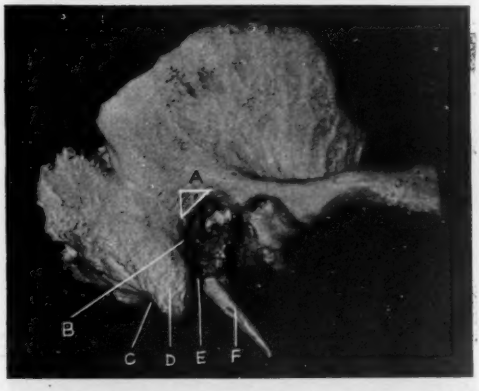


Figure 2. Lateral view of right temporal bone. A, Suprameatal triangle of Macewen. B, Tympanomastoid fissure. C, Digastric groove. D, Mastoid process. E, Stylomastoid foramen. F, Styloid process.

ing the function of the floor of the middle fossa of the skull and the roof of the epitympanicum and antrum tympanicum.

Thirdly, after the mastoid cavity is opened we take into consideration the posterior superior wall of the meatus through which the aqueductus Fallopii descends from a point on the inner wall of the antrum through the petromastoid portion of the temporal bone to its termination at the stylo mastoid foramen just posterior to the styloid process.

Gleason states "the aqueductus is a tube of ivory-like bone practically a part of or continuation of the capsule of the labyrinth."

Fourthly, we must consider the pronounced bulging seen on the inner wall of the mastoid cavity produced by the sigmoid groove lodging the lateral sinus.

Now that the operator has these anatomical landmarks to guide him in his operation, as soon as he has laid open the mastoid cells he endeavors to locate the antrum tympanicum in order to establish a communication between the epitympanicum and the mastoid cavity proper through the antrum, and when he has established a passage through these two cavities he knows at once his superior location of the descending portion of the aqueductus Fallopii and he feels a little more at ease, knowing that he is in the antrum. But does he feel as safe now that he is ready to proceed and convert his simple mastoid operation into a radical mastoid operation by cutting away the superior and posterior portion of the external auditory canal as represented by the horizontal and descending por-

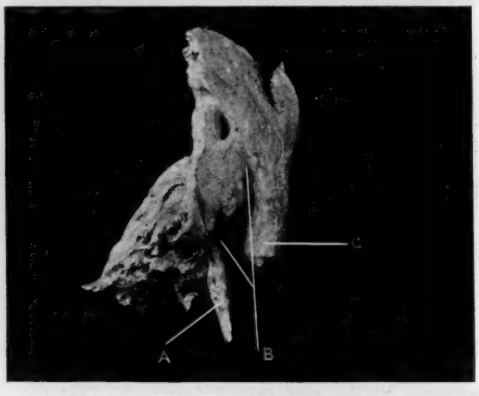


Figure 3. Posterior view of right temporal bone. A, Styloid process with stylomastoid foramen immediately behind. B, Digastric groove leading from behind forward to stylomastoid foramen. C, Mastoid process.

tions of the squamous and its articulation with the tympanic process of the temporal bone? No he does not, because he does not know the line in the curetted and cleaned-out mastoid cavity that would correspond to the line of the descending portion of the aqueductus Fallopii below the point at its turn on the inner wall of the antrum to its termination at the stylomastoid foramen. Therefore, not being fortified by this line he does not feel as free for the reason that the facial nerve is always in danger of injury.

While making a series of specimens of the macerated bone—knowing that in every case the digastric fossa, which is more or less deep, leads invariably from behind forward directly to the stylomastoid foramen, and on removing the cortex of the mastoid cavity and exenterating the cellular structure I found on the inside of the

mastoid cavity invariably a ridge corresponding to the digastric groove produced by the outward and downward growth of the mastoid process.

Now knowing that the digastric groove externally leads invariably to the stylomastoid foramen, it necessarily follows that the digastric ridge internally must lead to the stylomastoid foramen. Not only did I notice the ridge produced in the mastoid cavity by the digastric fossa, but running along the ridge from behind forward I usually found a partial lamina extending all along the line of the crest of the ridge up along the posterior wall of the external auditory canal corresponding to the line of the aqueductus Fallopii.

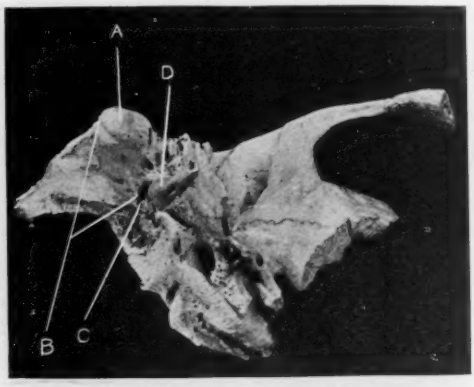


Figure 4. View of inferior surface of right temporal bone. A, Tip of mastoid process. B, digastric groove leading from behind forward to stylomastoid foramen. C, Stylomastoid foramen located in the petrous portion immediately behind styloid process. D, Styloid process joined to the petrous portion of the temporal bone at about birth.

Now what does this fact afford us? It aids us in accurately locating the line of the descending portion of the aqueductus Fallopii and the location of the stylomastoid foramen while working in the mastoid cavity, and we can work with more freedom in removing the posterior and superior wall of the external auditory canal, as we have the line directly in front of us that corresponds to the line of the aqueductus Fallopii from its turn on the inner wall of the antrum to its termination at the stylomastoid foramen.

Again it may be impossible to curette out the cells between the inner wall of the ridge and the sigmoid fossa due to the fossa impinging greatly on the mastoid cavity; in that case all that is necessary is to clean off the external surface or slope of the ridge from the bottom to the crest and from behind forward to the premastoid

lamina or posterior wall of the external auditory canal, and we get the same result; namely, the exact location of the stylomastoid foramen and the line of the descending portion of the aqueductus Fallopii.

Again, if the operator, after having opened the antrum, commences at the tip of the mastoid to curette up along the external slope of the digastric ridge his chances of penetrating the sigmoid fossa and injuring the lateral sinus are lessened, as I always found an area of cellular structure more or less between the inner wall or slope of the digastric ridge and the sigmoid fossa.

In conclusion, therefore, having a point in the mastoid cavity at

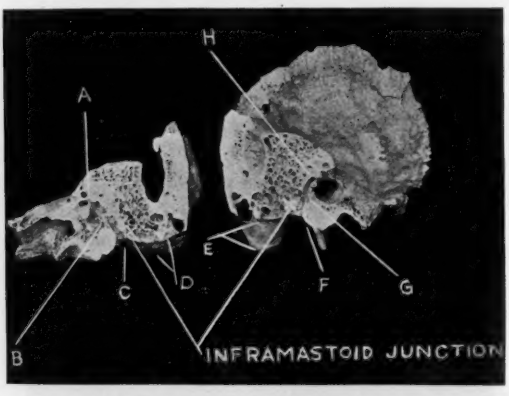


Figure 5. Left temporal bone. Sagittal section thru roof of digastric groove and thru facial canal. A, Beginning of the descending portion of the aqueductus Fallopii as it passes between the fenestra ovalis and the external semicircular canal. B, Aqueductus Fallopii. C, Stylomastoid foramen. D, Showing height of inner wall of digastric groove. E, Showing height of outer wall of digastric groove. F, Stylomastoid foramen. G, Aqueductus Fallopii. H, Beginning of the descending portion of the aqueductus Fallopii. (Inframastoid junction.)

the junction of the ridge with the posterior wall of the external auditory canal, which point internally corresponds to the location of the stylomastoid foramen externally, I propose naming this point the *inframastoid junction.* And a line run from the inner wall of the antrum to the inframastoid junction maps out the line of the facial canal from its turn on the inner wall of the antrum to its termination at the stylomastoid foramen.

RESUME OF DISCOURSE ON LANTERN SLIDES.

The first picture is a view of the left temporal bone of the newborn. The temporal bone is made up of three genetically distinct portions, namely, the squamous, the petromastoid and the tympanic.

At this age the mastoid portion of the petromastoid is undeveloped as yet and the mastoid tubercle "D" is the only suggestion we have of it, and this is found on the base of the petrous portion posterior to and slightly above the horizontal diameter of the "A" annulus tympanicus.

The mastoid tubercle grows downward and outward by its own growth and partly by the action of the sternocleido mastoid muscle and it reaches its adult form in the third year of life, during which time the digastric groove is developing. The "B" styloid porcess is found in the fissure between the annulus tympanicus and the mastoid tubercle and immediately behind is seen the "C" stylomastoid foramen. The petro squamosal suture is seen at "E," which, with

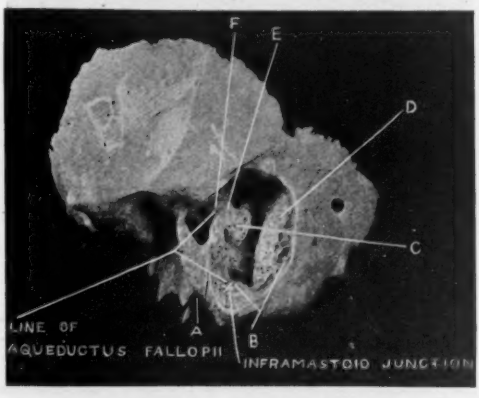


Figure 6. Left temporal bone. A, Stylomastoid foramen. B, Showing external slope and crest of digastric ridge. C, Posterior semi-circular canal. D, Sigmoid groove. E, External semicircular canal. F, The beginning of the descending portion of the aqueductus Fallopii. (Line of aquaductus Fallopii.) (Inframastoid junction.)

the development of the mastoid process, becomes the squamomastoid suture.

The second picture shows the right adult temporal bone. At "A" is seen the suprameatal triangle of Macewen, the boundaries of which are as follows: first, a line corresponding to the posterior root of the zygoma; secondly, a line passing through the quadrant formed by the superior and posterior portions of the external auditory meatus; thirdly, a line dropped vertically from the former passing through the latter at the posterior border of the external auditory meatus, an area externally corresponding to the location of the antrum tympanicum internally. "C" shows the digastric

groove running from behind forward, and at "D" is seen the mastoid process. The "F" styloid process is formed by the second and third segments of the hyoid bar of the second visceral arch and is found in the "B" tympanomastoid fissure and immediately posterior is the "E" stylomastoid foramen, located in the petrous portion of the temporal bone, showing that the location of the stylomastoid foramen, together with the projection of the descending portion of the aqueductus Fallopii, cannot vary greatly in the sagittal plane posterior to the tympanomastoid fissure, but that the location of the stylomastoid foramen, together with the projection of the descending portion of the aqueductus Fallopii, can vary greatly in the

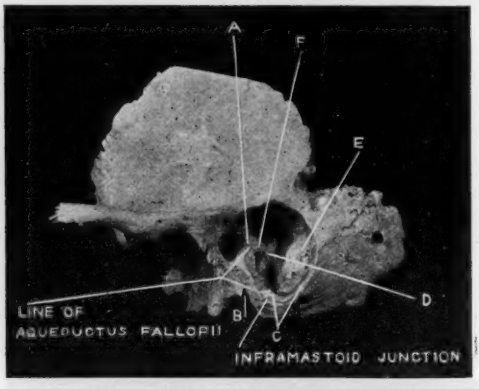


Figure 7. Left temporal bone. A, Beginning of the descending portion of aqueductus Fallopii. B, Stylomastoid foramen. C, Showing external slope and crest of the digastric ridge. D, Posterior semicircular canal. E, Sigmoid fossa. F, External semicircular canal. (Line of aqueductus Fallopii.) (Inframastoid junction.)

coronal plane external or internal to a vertical line passing through the beginning of the descending portion of the aqueductus Fallopii on the inner wall of the antrum as seen in the different types of skulls, namely, the long, medium and short.

The third picture is a posterior view of the right temporal bone, showing "A" styloid process; "B" the digastric groove leading from behind forward to the stylomastoid foramen immediately behind the styloid process. Note the height of the external wall of the digastric groove from the tip of the mastoid process "C" up to the roof of the digastric groove.

The fourth picture is a view of the inferior surface of the right temporal bone showing the "B" digastric groove leading to the "C"

stylomastoid foramen immediately behind the "D" styloid process, located in the tympanomastoid fissure.

Picture five shows a sagittal section of the left temporal bone through the roof of the digastric groove and the "B" descending portion of the aqueductus Fallopii. Here is seen the inframastoid junction.

The sixth picture is a view in which a radical mastoid operation has been done. Here is seen the line of the descending portion of the aqueductus Fallopii which is located in the anterior wall of the mastoid cavity from a point on the inner wall of the antrum to the inframastoid junction, a point where the crest of the ridge "B" in-

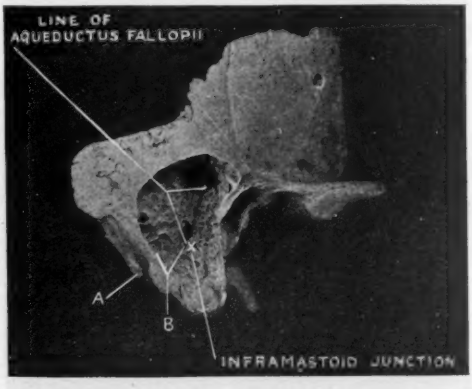


Figure 8. Right temporal bone. A, Digastric groove. B, Showing external slope and crest of digastric ridge. (Line of aqueductus Fallopii.) (Inframastoid junction.)

ternally, corresponding to the roof of the digastric groove externally, meets with the descending portion of the aqueductus Fallopii at the stylomastoid foramen. The external slope of digastric ridge is pronounced.

The seventh picture is a view in which a radical mastoid operation has been done. The line of the descending portion of the aqueductus Fallopii is seen on the anterior wall of the exenterated mastoid cavity from the inner wall of the antrum to the inframastoid junction. The "C" crest and external slope of the digastric ridge is seen.

The eighth picture is a view in which the simple mastoid operation has been done. The "A" digastric groove leads from behind forward to the stylomastoid foramen, a groove externally corresponding to the digastric ridge internally. The "B" crest and ex-

ternal slope of the digastric ridge is seen. In doing the mastoid operation, the antrum having been opened, the operator should curette up along the external slope of digastric ridge from the tip to the crest.

The ninth picture shows the exenterated cavity of a simple mastoid operation. Here is seen "A" digastric groove externally corresponding to "B" digastric ridge internally. Note the space between the crest of the digastric ridge and the "C" sigmoid fossa. The line of the aqueductus Fallopii is seen on the anterior wall of the mastoid cavity from the inner wall of the antrum to the inframastoid junction.

A view of the tenth picture shows a coronal section through tip of mastoid process, a simple mastoid operation having been done.

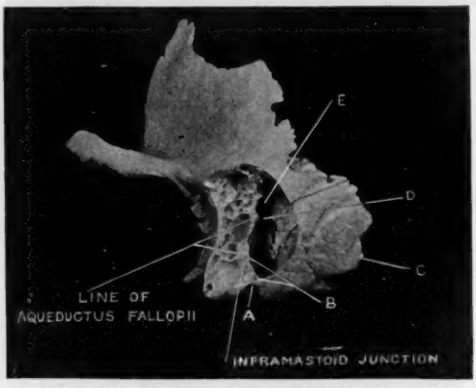


Figure 9. Left temporal bone. A, Digastric groove. B, Showing crest and external slope of digastric ridge. C, Sigmoid groove. (Note distance between crest of digastric ridge and sigmoid groove.) D, Beginning of descending portion of aqueductus Fallopii. E, Antrum tympanicum. (Line of aqueductus Fallopii.) (Inframastoid junction.)

Here is shown the line of the descending portion of the aqueductus Fallopii on the anterior wall of the exenterated mastoid cavity from a point on the inner wall of the antrum to the inframastoid junction, a point internally corresponding to the stylomastoid foramen externally. "B" is compact cortical bone, constituting the external wall of the digastric groove and the outer slope of the digastric ridge internally. "C" constitutes the roof of the digastric groove externally and the crest of the digastric ridge internally. "D" constitutes the internal wall of the digastric groove externally and the internal slope of the digastric ridge internally. Note the distance or

space between the crest and internal slope of the digastric ridge and the wall of the sigmoid fossa through which the section passes.

The measurements of the digastric groove and the descending portion of the aqueductus Fallopii as found on the different types

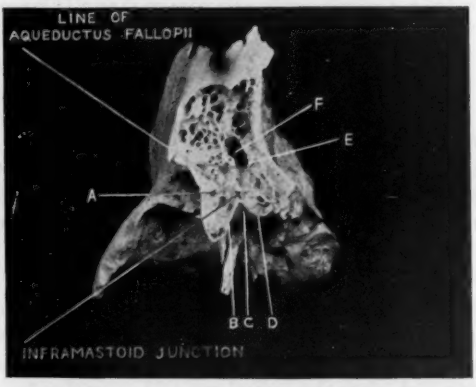


Figure 10. Left temporal bone. Frontal section through middle of exenterated mastoid. A, Posterior wall of external auditory canal. B, External slope of digastric ridge. C, Roof of digastric groove externally which produces crest of digastric ridge internally. D, Internal slope of digastric ridge. (Note distance between internal slope of digastric ridge and sigmoid groove.) E, Beginning of descending portion of aqueductus Fallopii. F, Antrum tympanicum. (Line of aqueductus Fallopii.) (Inframastoid junction.)

of skulls, namely, the brachycephalic or short skull, whose index is above 80; the mesaticephalic, whose index is between 75 and 80, and the dolichocephalic or long skull, whose index is below 75, will be presented in a later report.

3332 Chestnut Street.

TOTAL DEAFNESS DUE TO TRAUMA, WITH NORMAL STATIC LABYRINTHINE FINDINGS. REPORT OF TWO CASES*

DR. SAMUEL J. KOPETZKY, DR. ALFRED A. SCHWARTZ,
New York City.

Complete destruction of the internal ear, whether due to trauma or disease, has been frequently reported, and two such cases (one of which appeared after a radical mastoidectomy, and the other due to a chronic suppurative process) have been recently reported by the writers.

Because of the peculiar anatomy of the membranous labyrinth, it is not strange that the entire organ should be destroyed when part of it is attacked by a suppurative process. The cases of circumscribed labyrinthitis reported deal with necrosis of part of the bony capsule, and when the radical mastoid operation has been performed, the symptoms due to the disease usually disappear. When a diffuse purulent labyrinthitis sets in, the diagnosis is based upon the appearance of total deafness in addition to the labyrinthine symptoms; and, according to Friesner and Braun, and others, if hearing persists, this diagnosis is excluded.

The perilymph channels of the static labyrinth are continuous with those of the cochlea, through the scala vestibuli, the latter communicating with the scala tympani through the helicotrema. The endolymph channels of the static labyrinth communicate with the essential portion of the cochlea, the ductus cochlearis, through the canalis reuniens. The membranous labyrinth is enclosed in a firm bony capsule of small caliber, and a suppurative process would hardly destroy one portion of the internal ear and leave the other functionating. Theoretically, it is possible; but we have been unable to find an authoritative case of such a condition in the literature.

That the cochlea may be non-functionating, and the static labyrinth be intact, is well known, and needs only passing comment. For cases of deaf-mutism with normal vestibular reactions are not infrequent; and at the present time we have under observation a case where there is total deafness in one ear, due to a non-suppurative disease in childhood (probably a meningitis), and whose static labyrinth not only responds, but is apparently hypersensitive. In

*Read before the Section on Otology, New York Academy of Medicine, April 14, 1922.

this case the drum is absolutely normal, and the Eustachian tubes patent.

There have been cases reported of total deafness due to trauma, where the static labyrinth has been found normal—notably, one by Auerbach in 1913 and two by M. J. Gottlieb in 1921. And it is because of the importance of the legal aspect of this question that we desire to add to the literature the two cases which follow.

Total unilateral deafness is the most frequent form of otologic malingering; and if it be true that traumatic destruction of the auditory end organ can take place without involving the remainder of the internal ear, then the Barany tests cannot be relied upon to show even in all probability that the entire labyrinth is functioning because the static portion responds. Of course, a non-reacting static labyrinth supports a claim of complete destruction of the power of hearing; but the cases quoted above and the two here presented prove conclusively that the presence of a normally reacting labyrinth does not rule out a completely non-functionating cochlea.

Total traumatic deafness with normal static reactions may, of course, be central in origin. Oppenheim believes that the lesion in these cases is located in central nuclei of the eighth nerve. On account of the relations of the two auditory nuclei in the medulla it is hardly probable that destruction of these could take place in this locality without severely injuring or destroying other paths and nuclei which lie about them. This same holds true for the pathways from the dorsal and ventral nuclei; and it is only when the auditory centre in the superior temporal gyrus is considered—or the junction of the tracts leading to it from the postgeminum and postgeniculum —that a central causation of the destruction of the auditory fibres can be seriously discussed.

In an histological study of the labyrinths in cases where there had been severe cranial injuries, Barnick was able to demonstrate extravasations of blood into the maculae acousticae and into the vestibular end plates. Politzer and Gradenigo are of the opinion that the otitic symptoms in cranial injury are due to hemorrhages into the labyrinth—especially into the cochlea; and this view finds support in the experiments of Lange, who repeatedly found hemorrhages in the labyrinths after severe cranial injuries to rabbits.

Case 1: D. S., age 27, first came under observation on November 5, 1921. He was a veteran of the British Army, and had been in active service over five years. The only injury sustained in all that period was what he termed "a splinter" of shell in the cheek, which had been easily removed. He denies ever having been "shell shocked" and denies venereal disease.

On April 26, 1921, he was struck by an automobile, and was unconscious for five days. Immediately after the accident, he was removed to the Metropolitan Hospital, where he remained for two months.

Prior to the accident the patient claims that he could hear perfectly well; that there had never been any tinnitus, and that at no time had there been an aural discharge. He had never complained of nasal trouble and there had been no attacks of sore throat.

As soon as he regained consciousness (five days after the accident), he realized that he could not hear at all with his left ear. Hearing in the right ear seemed poor, and from then on appeared to gradually become worse.

There was bleeding from the interior of the canal of the left ear for three days after he regained consciousness, and then the discharge became purulent. This discharge gradually lessened, and three weeks before coming to us, had stopped completely.

There was no discharge from the right ear at any time, but from the onset he complained bitterly of a constant tinnitus in this ear, of a loud drumming character, which has persisted up to the present time.

When he became conscious, marked vertigo was present—the room seemed to turn about him; he does not know in which direction. He was compelled to lie on his back because of bandages, etc., and does not know if lying on one side or the other might have brought relief. He remained in bed three weeks. After he was about, he had a staggering gait, but does not know if he veered to right or left. Lately he has noticed that he always veers to the right when walking.

He has had attacks of vertigo about three times a week since the accident. When they come on, objects seem to rise upward, or turn around him. There is no nausea nor vomiting. At times he falls and becomes unconscious, and remains so for several hours. These spells of unconsciousness have been becoming more frequent of late. He has hurt himself slightly at times when he fell. There has been no frothing at the mouth and he has never bitten his tongue, or shown other symptoms of what might be considered a true epilepsy. When standing still, he thinks that he is falling forward. He becomes dizzy when bending over.

The last attack of vertigo occurred four days ago, while he was in bed. He became unconscious as in the other attacks, and came out of it feeling perfectly well.

He sees well with both eyes. He complains of continual headaches, mainly in the left parietal region.

Otoscopic Examination: Right Ear: Nick in rim of concha at upper anterior angle. Scar on drum, thickness and retraction. Left Ear: Scar on concha on helix, upper portion of same, extending backward to rim. Loss of cartilage in helix, upper and external half gone. Scar and thickness and retraction of drum, more marked than on right side.

Functional Tests: Lateralization: To right. *Right*: C fork(air), 12; C fork (mastoid) 8; C_4, 9; 64, D. V., not heard. *Left*: C fork (air) 0; C fork (mastoid) 0; C_4, 7; 64, D. V., not heard. With noise apparatus in right ear, does not hear in left ear. With noise apparatus in left ear, hears in right ear.

Neurological and Spontaneous Vestibular Reactions: Is unsteady when standing on one foot. Spontaneous nystagmus: None. Spontaneous pastpointing: To right with right hand 2″ to 6″; to right with left hand 2″ to 6″. Walking: Very slight veering to right—usually walks in straight line. Romberg: Sensation of falling forward; slight swaying forward; stands with trunk bent forward. Ataxia (upper extremities): None—finger to finger and finger to nose test normal. Pupils: Equal, react to light and accommodation. Visual fields: apparently somewhat contracted when hand is below. Pelvic Girdle: Normal. Reflexes: Very active; much more marked on right side. Tremor: Marked tremor, both hands, coarse.

Vestibular Tests: To Test Horizontal Semi-circular Canals: Turning to right: Nystagmus: 34 sec. to left, large amplitude, horizontal. Vertigo: 22 sec. to left. Pastpointing: Normal, both hands to right. Turning to left: Nystagmus: 30 sec. to right, amplitude smaller than on turning to right. Vertigo: 20 sec. to right. Pastpointing: Normal with right hand to left. (Pastpoints 6 to 8 inches to right with left hand. Repeats exactly on check test.)

To Test Vertical Semi-circular Canals: Turning to right; Nystagmus: 31 sec. to left, rotary. Wide amplitude. Vertigo: 24 sec. to left. Falling: Normal to right; sensation of falling to left. Turning to left: Nystagmus: 28 sec. to right, rotary, wide amplitude. Vertigo: 20 sec. to right. Falling: Normal to left, sensation of falling to right.

Caloric Tests, Irrigating with Cold Water: Irrigating left ear, to test left vertical canals: Nystagmus: Marked, rotary, to right, after 22 seconds (patient became very ill). Vertigo: Normal, marked. Pastpointing: Normal, both hands to left. Falling: Head back to test left horizontal canal: Nystagmus: Normal, active, horizontal to right. Pastpointing: Normal.

Summary: 1. The patient is totally deaf in the left ear.

2. The static labyrinth of the deaf ear responds promptly to caloric stimulation, and the reactions are normal.

3. There is spontaneous pastpointing with both right and left hands; he is unsteady when standing, and with his eyes closed, has a sensation of falling forward. There is a marked tremor present, but there are no definite cerebellar or cerebral symptoms present which would localize a central lesion.

4. There is abnormal pastpointing with the left hand on turning to the left, to test the horizontal semicicular canals. The pastpointing was to the right, and was repeated exactly on a check test.

5. All the other reactions were within normal limits.

Conclusions.

1. Although the spontaneous pastpointing, and the atypical pastpointing in one of the tests, may point to a central lesion, there was no confirmation in the other tests (namely, the caloric or the rotation tests to test the vertical canals). Whether there is some central lesion, is therefore undecided.

2. There is a total deafness of the auditory portion of the eighth nerve, with an actively functionating labyrinth.

3. The onset was traumatic, the deafness coming on immediately after injury—or at least being noticed as soon as the patient became conscious.

4. At the onset labyrinthine symptoms were presented, which gradually cleared. This may have been due to irritation of the static labyrinth, occurring at the same time as the destruction of the auditory portion. Whether there is still some condition present in the labyrinth which causes this irritation is problematical, for there is a possibility that the vertiginous attacks which the patient is now having are of central origin.

We are led to conclude that the case is one of traumatic destruction of the cochlea itself, due to hemorrhage, the static labyrinth being intact.

Case 2: E. L., age 20, first came under observation on February 25, 1922.

Seven years ago, he was knocked down and run over, and the entire left side of the body injured. He was not unconscious, and after the injury immediately got up, but was so "dizzy" that he fell. He does not remember whether this was a true vertigo—whether external objects moved about him, or whether he himself turned—but he states definitely that, after the first day of his illness, there was at no time vertigo, nausea or vomiting. He was

confined to bed for eight weeks because of his injuries, but all of these cleared completely except that to his hearing.

Prior to the accident, the patient claims that he could hear perfectly well. Immediately after, he was completely deaf in the left ear. At no time was there tinnitus, pain or discharge.

This deafness has persisted unchanged up to the present. The patient feels absolutely well, has never had vertigo or other aural symptoms, and came to us to see if there was any possibility of improving the hearing.

Otoscopic Examination: Right ear: Slight retraction of drum, no scars. Left ear: Normal in appearance. Functional Tests: Lateralization: Right: C fork (air), 32; C fork (mastoid), 20; C_4, 34; 64 D. V., heard. Left: C fork (air), 0; C fork (mastoid), 12 (to right side); C_4, 0; 64 D. V., not heard. With noise apparatus in right ear, does not hear in left. With noise apparatus in left ear, hears in right.

Neurological and Spontaneous Vestibular Reactions: Spontaneous nystagmus: None. Spontaneous pastpointing: None. Walking: Walks in straight line. Romberg: None. Ataxia (upper extremities): None. Pupils: Equal, react to light and accommodation. Visual fields: Normal (apparently). Pelvic girdle: Normal. Reflexes: Very active—equal.

Vestibular Tests: To Test Horizontal Semicircular Canals: Turning to right: Nystagmus: 32 sec. to left, active, short, horizontal. Vertigo: 13 sec. to left. Pastpointing: Pastpointed once with right hand to right, touched with left hand, then touched with right hand. Turning to left: Nystagmus: 25 sec. to right—active, wide, horizontal. Vertigo: 10 sec. to right. Pastpointing: Touched exactly, both hands.

To Test Vertical Semicircular Canals: Turning to right: Nystagmus: 15 sec.—slight rotary—to left. Vertigo: 8 sec. to left. Pastpointing: Normal, both hands to right. Falling: To right, slight. Claims sensation of falling to right. Turning to left: Nystagmus: 20 sec. to right, moderate, rotary. Vertigo: 8 sec. to right. Pastpointing: Normal, both hands to left. Falling: Normal to left—marked sensation of falling to right.

Caloric Tests, Irrigating with Cold Water: Irrigating right ear, to test right vertical canals: Nystagmus: After 35 sec., normal rotary, marked, to left. Vertigo: Normal. Pastpointing: Touched exactly, both hands. Falling: Head back to test right horizontal canal: Nystagmus: Normal; marked to left, horizontal. Pastpointing: Normal with right, touched with left. Irrigating left ear to

test left vertical canals: Nystagmus: After 40 sec., normal, marked rotary, to right. Vertigo: Normal, marked (vomited). Pastpointing: Slight—both hands normal to left. Falling: Head back to test left horizontal canal: Nystagmus: Normal, large, horizontal, to right. Pastpointing: Normal, both hands to left.

Summary.

1. The examination of the auditory part of the eighth nerve shows a complete deafness in the left ear. The hearing in the right ear is reduced to about ⅔ of normal.

2. The examination of the static labyrinth and its tracts showed absolutely normal reactions, with the exception of pastpointing in testing the horizontal canals. The other tests (caloric) showed normal pastpointing.

Comment.

1. There is, in this case, total deafness with a normally reacting static labyrinth. The loss of hearing came on immediately after trauma, and is complete.

2. In this case there was no question of compensation for the injury and no legal problem. The patient, in perfect general condition, came simply to find relief for his deafness.

Conclusions.

1. Complete deafness may occur without involvement of the static labyrinth or its tracts. In Case II there were no labyrinthine symptoms whatever after the first day of the disease, and the vestibular reactions are now normal. In the first case, there is total deafness with a reacting labyrinth; but there is also evidence of other injury.

2. It seems improbable to us from a study of Case II that the lesion is of central origin. Although there is no definite proof of such being the case, it seems more likely that the injury caused a destruction of the end organs, by destruction of the cochlea.

3. These cases are reported because of the injustice which may be done to patients who are suspected of malingering deafness, and whose vestibular reactions are normal. In Case I, the patient was suing because of the accident and all the tests for malingering at our disposal were resorted to. We were absolutely convinced that he was totally deaf in the affected ear, even though the static labyrinth reacted normally.

51 West 73rd Street.

PRIMARY THROMBOSIS OF THE MASTOID EMISSARY VEIN WITH SECONDARY INVOLVEMENT OF THE LATERAL SINUS.*

DR. JOSEPH FRIEDMAN AND DR. SAMUEL D. GREENFIELD,
Brooklyn, N. Y.

Ordinarily the mastoid emissary vein is an insignificant structure. It is encountered during the mastoid operation calling the surgeon's attention to its existence only as a result of the annoying bleeding that oftentimes is occasioned by its injury. The mastoid emissary vein emerges from the mastoid foramen. It collects the blood from the soft parts over the mastoid region and anastomosis with the occipital vein at its exit from the foramen.

The location of the mastoid foramen is exceedingly variable, but in the majority of instances it is found at the posterior extremity of the occipital groove, which corresponds to a point one-half inch above and the same distance behind the center of the mastoid tip. The vein after entering the foramen passes forward a variable distance through the mastoid process until it reaches the sigmoid portion of the lateral sinus into which it empties.

It can therefore readily be understood that in cases where there are diseased cells encountered along the course of the vein the latter may easily become implicated as a result of direct extension of the infectious process. In those cases in which there are cells located behind the sinus, and especially if the disease has existed for some period, this is more apt to occur. It is upon involvement of these posterior cells that one elicits the so-called tenderness over the emissary region, under which circumstances, however, it does not necessarily imply that the emissary itself is involved. According to Franchere, the elicitation of distinct tenderness over the emissary is conclusive evidence of the presence of a severe inflammation of the mastoid and furthermore it signifies that this patient must eventually come to operation. However, this is not invariably true, for we have seen cases in which there was distinct tenderness over this area and yet these patients have recovered spontaneously.

Griesinger was the first to call attention to the edema and swelling over the emissary region in connection with thrombosis of the lateral sinus. As a result of obstruction to the return flow of blood in the lateral sinus there is backing up in the emissary with the pro-

*Taken from the Oto-Laryngological Service of the Beth Moses Hospital.

duction of edema and infiltration of the tissues about the vein. More often, however, the emissary itself is obstructed by an obturating thrombus which has its origin in the lateral sinus and through extension involves the mastoid emissary. Thus in most instances involvement of the emissary vein is to be considered secondary. Nevertheless it is reasonable to suppose that this structure may become implicated primarily, and there is no doubt in our minds that the emissary is infected and obstructed in many more instances than we have been led to believe, but because of the process remaining essentially local there have been few or no manifestations pointing to the presence of such a condition.

We believe that the emissary, especially if of unusual size, may become infected primarily and as a result give systemic manifestations similar to those occasioned by thrombosis of the lateral sinus itself. Furthermore we are of the opinion that the mastoid emissary vein when infected under these circumstances through a process of extension may give rise secondarily to infection of the lateral sinus with a typical clinical picture, and with a characteristic course.

The case we are reporting falls into the category just described, in which there was originally a badly infected mastoid, with involvement of the mastoid emissary, giving rise to typical systemic manifestations with secondary implication of the lateral sinus.

"Report of Case." M. T., aged ten, was taken ill on January 5, 1922, with severe pain in his right ear. The pain persisted for three days, at which time we saw the patient and found a bulging drum and a moderate amount of mastoid tenderness. We incised and obtained pus. The otitis ran the usual course for several days, the ear discharging profusely, but the mastoid tenderness persisted and the patient complained of spontaneous pain at night. On the sixth day of his illness there appeared some edema over the emissary region associated with exquisite tenderness. For several days following the patient improved somewhat; the otoscopic picture was good; the mastoid tenderness seemed to disappear excepting that located at the area of edema, over the posterior of the mastoid. The temperature on the eighth day of his illness began to remit, rising in the evening to 104°, and dropping to normal in the morning. There were no chills nor sweats, urine was negative, heart and lungs were normal. We recommended the hospital for observation. On January 15, the patient was admitted to the Beth Moses Hospital with a diagnosis of acute mastoiditis and probable sinus thrombosis.

January 16, 1922. Patient's condition remained unchanged. The

edema over the emissary was somewhat increased and the tenderness persisted. Urine examination was negative. Blood count showed 11,200 WBC with 79 per cent polys. Blood culture taken was reported sterile at the end of twenty-four hours. X-ray of both mastoids taken.

January 17, 1922. Widal reported negative (dilutions 1:20—1:60—1:20). Blood culture reported sterile after forty-eight hours. X-ray report by Dr. Bernstein. Right mastoid shows obliteration of cell outlines. Diagnosis—Right Mastoiditis. The temperature that evening rose again to 104.2, and we decided to operate.

January 18, 1922. Mastoid operation. The findings at operation were as follows: the mastoid was a large, pneumatic one; the cells were filled with profusely bleeding granulations; some free pus was present in the antral region; culture subsequently showed the streptococcus. The cells were thoroughly exenterated and the entire mastoid dissected. The lateral sinus was uncovered from knee to tip and found to be perfectly normal. The most interesting feature at the operation was the unusually large mastoid emissary vein. It was intact and could be traced from its entrance through the cortex to its junction with the lateral sinus. The diameter of the vein was at least three-eighths of an inch; it was discolored, black and thrombosed, but the disease seemed to stop short of the lateral sinus, the latter structure being seemingly unaffected. It was the largest emissary we have ever seen. We decided not to disturb the vein; the wound was packed with iodoform and left widely open.

January 19, 1922. The patient rallied from the operation, but the temperature remained unaffected. That same evening there was the usual rise to 105°.

January 20, 1922. The patient's condition was fair. He complained of headache and seemed to be somewhat drowsy; the blood culture taken was subsequently reported sterile after twenty-four hours; white blood count showed 12,800, polys 78 per cent.

January 21, 1922. The first dressing was performed under gas anesthesia and the wound was carefully inspected. The emissary was necrotic and there was plainly visible a line of separation between it and the sinus, as though there were an attempt made for it to slough off. Dr. Friesner was present at the dressing and he concurred in the procedure we had followed at the operation.

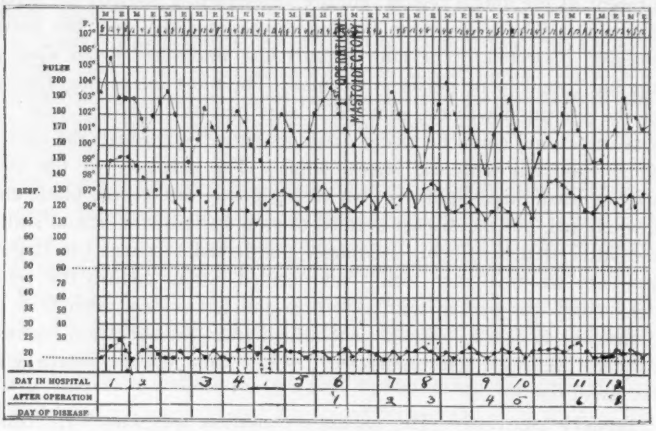
January 22, 1922. The temperature continued to remit, the patient presented the classical euphoric aspect. There were no chills nor sweats. Dr. Gingold's examination revealed nothing abnormal. He referred the patient back to the otologist.

January 23, 1922. Blood count showed WBC. 18,000, polys 87 per cent; blood culture taken was reported negative; spinal tap by Dr. Bender 15cc. under slight pressure obtained; fluid clear; cytology 10 cells per cmm; bacteriology negative; chemistry negative.

January 24, 1922. Neurological examination by Dr. Wechsler negative for meningitis or other intracranial complications. Everything pointed to a sinus condition.

January 25, 1922. Patient seemed to lose ground. Anemia was increasing and evidences of sepis were apparent. We advised ligation of the jugular and exploration of the lateral sinus.

January 26, 1922. Jugular ligation and exploration of the lateral sinus. In view of the fact that the sinus had been well uncovered



at the primary operation we went into the neck immediately. The internal jugular vein was uncovered and ligated below the facial. There was nothing abnormal noted about the vein. We now went into the mastoid wound and found the sinus wall much thickened, the emissary vein came away in one mass, there being no bleeding at the point of severance with the sinus. The latter was incised and no obturating thrombus was detected. There was free bleeding from the torcular and bulbar ends. The wound was packed with iodoform plugs, and hemorrhage controlled. The child was given an intraveanous while on the table and put on stimulation.

January 27, 1922. The patient rallied from the operation. The most interesting feature was the drop in temperature to 100°, and the absence of the usual rise in the evening. From this day on, as

can be seen in the accompanying temperature charts, the fever remained below 101°.

January 28, 1922, to February 8. The child quickly improved; slept soundly; ate well; speedily gained weight. The neck wound healed by primary intention and the mastoid wound was granulating and the patient was well on the way to recovery.

February 9, 1922. The patient passed a large amount of bloody urine. There were no accompanying symptoms, no pain, no rise in temperature. Examination revealed a high albumen content due probably to the contained blood with many casts. This continued for several days, but gradually subsided and on February 12, the

urine was clear with the exception of a slight trace of albumen and occasional casts.

February 14, 1922. The patient left the hospital and in six weeks from this date he was discharged from our care as cured.

This case presents a number of very interesting features to which we wish to call particular attention; first, the question of primary thrombosis of the mastoid emissary. As was stated before, primary thrombosis of the mastoid emissary vein may have its origin from infection of the mastoid cells, surrounding this structure. It may also follow upon infection and breaking down of post-auricular glands. We are of the opinion and there is no reason to doubt that this child's sepsis had its origin in the emissary vein. The fact that the temperature was of a septic nature very early in the disease, supplemented by the pathological findings at operation, we are jus-

tified in forming such deductions. True, the lateral sinus may be implicated without any external manifestations discernible at operation, yet it is reasonable to suppose that at least the early remissions in temperature resulted from involvement of the emissary vein. Fully two weeks elapsed from the time the septic temperature made its appearance to the exploration of the sinus, at which time very little disease was discovered, although from the clinical aspects we anticipated finding much more pathology in the latter structure. We do not lose sight of the fact, however, that these may be the sole findings where the sinus is the main source of trouble. We have all seen cases running a septic temperature with a negative blood culture, where ligation of the jugular is followed by recovery and in which one fails to find a thrombus in the sinus. We would therefore place this case on record as being one of primary thrombosis of the mastoid emissary.

Secondly, the immediate drop in temperature and improvement in the patient's condition upon ligation of the jugular. The blood stream evidently was being supplied with infectious material from the emissary and the lateral sinus in the same vicinity, and the cutting off of this source of infection brought about the quick change in his condition and cessation of his temperature.

Thirdly, the occurrence of a hematuria several weeks after the second operation. It is barely plausible to consider this as being metastatic in origin, resulting from the general infection of the blood stream. Too much time elapsed between the ligation of the jugular and cessation of the temperature to warrant such a presumption. The hematuria must be considered an accompanying symptom of an acute nephritis. Although there was an absence of much febrile reaction, edema, and scantiness of urine, examination of the latter disclosed the presence of some casts. According to Friesner an acute nephritis accompanying mastoid disease may present a picture of a similar nature, namely, with practically no systemic symptoms, but some urinary findings and a hematuria. As was stated before, the condition cleared up in several days.

691 Lafayette Avenue.

SENSIBILITY OF PATHOLOGICAL EARS TO SMALL DIFFERENCES OF LOUDNESS AND PITCH, INCLUDING A REPORT ON SEVEN CASES OF DIPLACUSIS.*

V. O. KNUDSEN AND DR. GEORGE E. SHAMBAUGH, Chicago.

Introduction—Dr. G. E. Shambaugh. This is a preliminary report of an investigation undertaken in the Department of Physics at the University of Chicago and which is of interest to otologists. It is an effort to determine the sensibility of the ear to small differences of loudness and pitch. Another investigation undertaken by Mr. Minton in the same laboratory has aimed to determine the minimum audibility of tones. The latter work is to be discussed at this meeting by Dr. Wilson. Both investigations have made use of the method of charting graphically the results which renders the study of their findings more simple.

The primary interest which these researches so far have had for the otologist is the side-light which they throw on the physiology of tone perception. Quite apart from this interest the question naturally arises whether the results of this work may not be found to be of some practical value in solving the clinical problems with which the otologist is confronted. Research is usually undertaken without a thought as to any practical application. Where such application is feasible this fact soon becomes apparent. These researches are not peculiar in that they do not aim to solve our clinical problems. Whether practical application can be made of these tests is a question for the otologist to work out. It is a question of no small importance. Any additional aid in our work is readily welcomed. The question is of special importance since there has been some discussion of a plan for establishing laboratories to which clinicians should send their patients to have these tests made and for which the patient should be charged a fee, somewhat as is being done in X-ray work. Such a plan, if carried out, should have the sanction of the otologist.

In order to be able to formulate a conclusion as to a practical application of these tests, it is of primary importance that we have a clear conception of our clinical problems, lest we be led into confusing a study which may be purely of scientific interest with something quite different. As regards the clinical problems involved in

*Read before the American Otological Society, May, 1922.

the hearing tests, the first question is concerned with distinguishing cases where there is some possibility of improvement by treatment from those where from our knowledge of pathology it is quite clear that treatment could be of no assistance. I do not believe that this diagnosis presents any particular difficulty for the experienced otologist. It is ordinarily rather easy to determine this by other simpler methods of examination. I have not been able to make out as yet that its solution is materially assisted by these additional tests. Still it is possible that something may yet be worked out along this line. Another practical use which is made of the functional tests of hearing is for determining what improvement, if any, is resulting from treatment of defective ears. This subject has received a good deal of attention in the past. The conclusion usually reached has been that the voice tests are superior to other methods, mainly because we are primarily interested in securing improvement in the hearing for the spoken voice and because it is well known that there need be no distinct relation between the ability to hear the voice and the hearing for some mechanical device, such as the watch and the various tuning forks. Tests showing the entire tone range should, it would seem, be able to afford some sort of an index of what we aim to get, that is, of the degree of improvement for the spoken voice. It does not seem probable, however, that the testing of the tone range would supplant the testing with the voice for this purpose unless some device should be invented which would render these tests simple enough to be carried out with sufficient readiness to serve this purpose practically.

It has been suggested that these tests are valuable when repeated from time to time for showing changing status of hearing in cases of progressive deafness, such as otosclerosis or primary nerve deafness. I am inclined to think that with these tests we can get a more satisfactory line on the progress of the hearing condition in these cases than by other methods. It is a question, however, whether we would be justified in advising patients to go through with these elaborate tests from time to time, especially since in just these cases there is no practical assistance to be derived from them.

There still exists a great deal of haziness in the minds of many practitioners regarding the clinical problems in otology. To assume that these newer tests of hearing are to bring in a new era in otology in which these tests will be able to dispel the prevailing pessimism that exists regarding the cure of deafness shows a failure to grasp the simple clinical problem presented in these cases. Pessimism does exist exactly as it does in the treatment of locomotor ataxia or

in cases where the hair is turning gray for the very good reason that we know from our study in pathology that such conditions as otosclerosis and degeneration of the eighth nerve can neither be improved nor their progress checked by treatment.

These remarks are intended as a warning against a too hasty conclusion on the part of otologists that these tests have a commercial

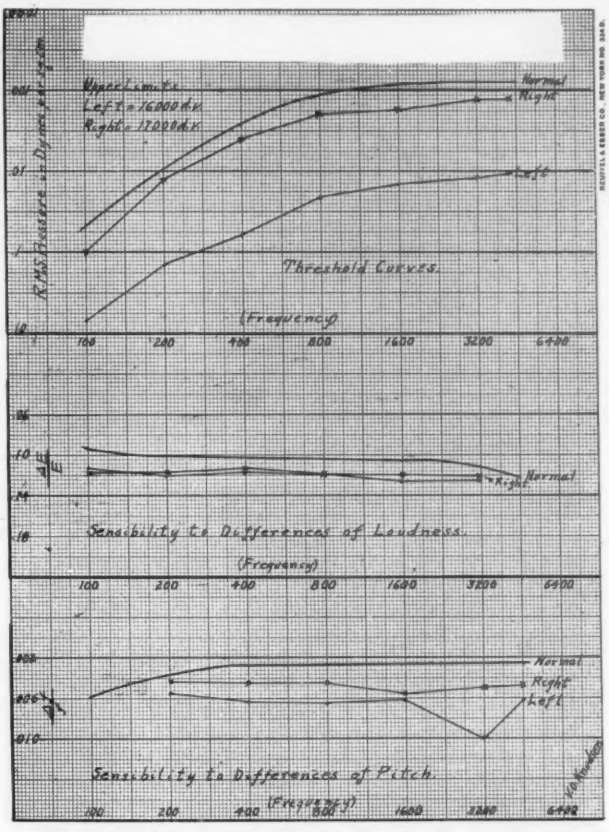


Figure A

value; in other words, are of practical assistance to the otologist. Since the time of Helmholtz the physicist has played an important part in the study of the problem of hearing. Much of this work has been done in co-operation with otologists. This co-operation, therefore, has long been established. It is to be hoped that something of practical value may come from the fresh impetus arising

from these newer tests. What we may expect quite probably will not be related to our primary problem, that of diagnosing the treatable from the incurable form of deafness or much less to dispelling existing pessimism regarding the incurable forms of deafness, but may rather be along the lines of improvements in the devices intended for bridging the chasm between defects in hearing and the nor-

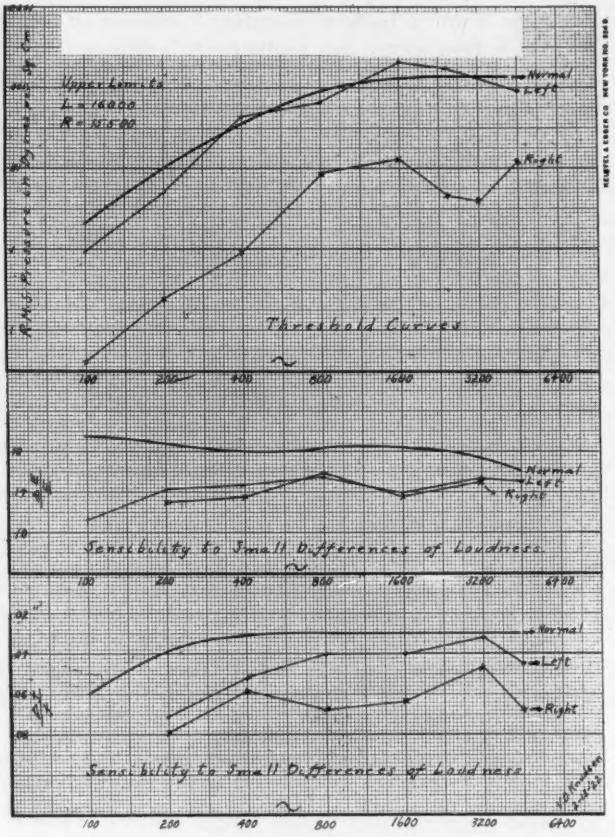


Figure B

mal ear. It would seem that a study of the minimum audibility of tones in the several parts of the tone range and of the sensibility of ears to small differences of loudness and pitch may furnish data which could be turned to a practical purpose in the construction of improved hearing devices, especially the electrical hearing devices,

which, imperfect as they now are, have already gained a wide application. Until it becomes apparent that these tests add something essential to our methods of diagnosis, something which simpler methods do not give, it would be a mistake to lend encouragement to the idea that this work has a practical value for which the patient can be asked to pay.

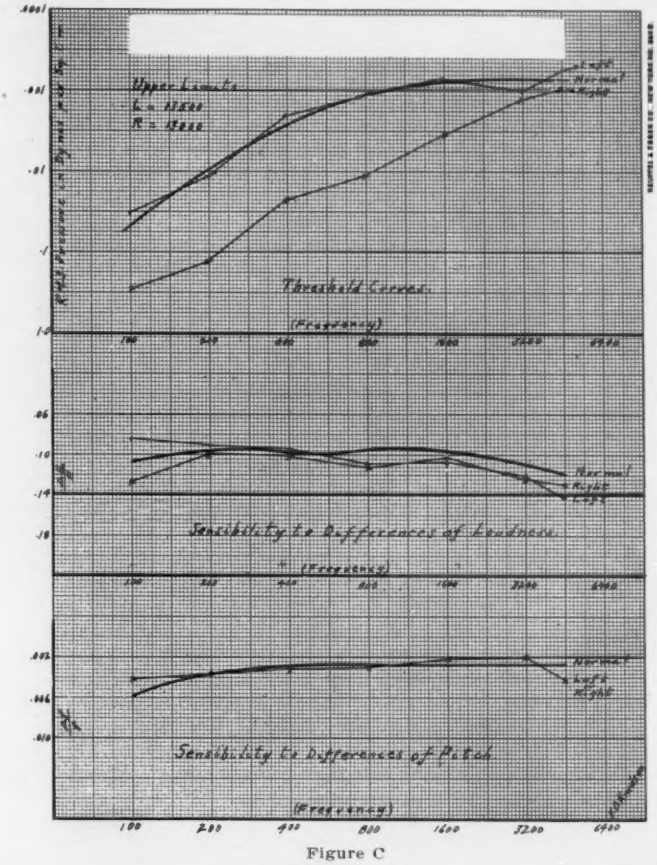


Figure C

The report of Mr. Knudsen follows: The writer has recently presented a paper before the American Physical Society on "The Sensibility of Normal Ears to Small Differences of Loudness and Pitch." The work reported in the present paper is an extension of that investigation applied to pathological ears. A knowledge of the

sensibility of the ear to small differences of loudness and pitch is essential to intelligently prescribe artificial aids for hearing. If a pathological ear is normal in its pitch and intensity discriminating power, appropriate amplification of the sounds coming to the ear will restore normal hearing. But, if the affected ear is seriously sub-normal for these functions, amplification is not adequate for

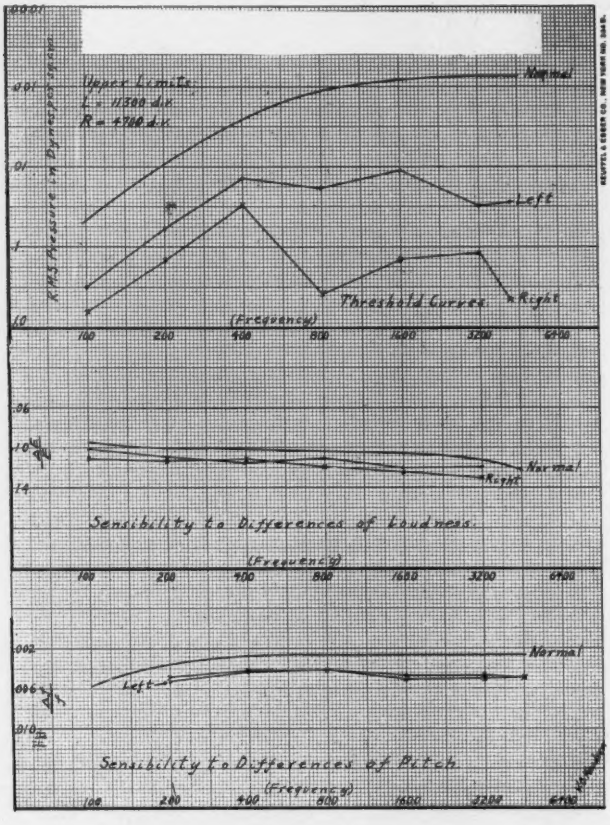


Figure D

the restoration of normal hearing, since the interpretation of speech and musical sounds by the ear requires the capacity to appreciate small differences of pitch and loudness.

The method of just discernible differences is employed in the present investigation for determining the sensibility of the ear to small differences of loudness and pitch. The source of sound used

is a telephone receiver actuated by energy from a vacuum tube oscillator. The oscillator will produce tones of any desired frequency between 30 d. v. and 20,000 d. v. By means of a divided resistance circuit the intensities of the tones can be varied by any desirable and measurable intervals from the threshold intensities up to intensities sufficiently loud to excite the sensation of pain.

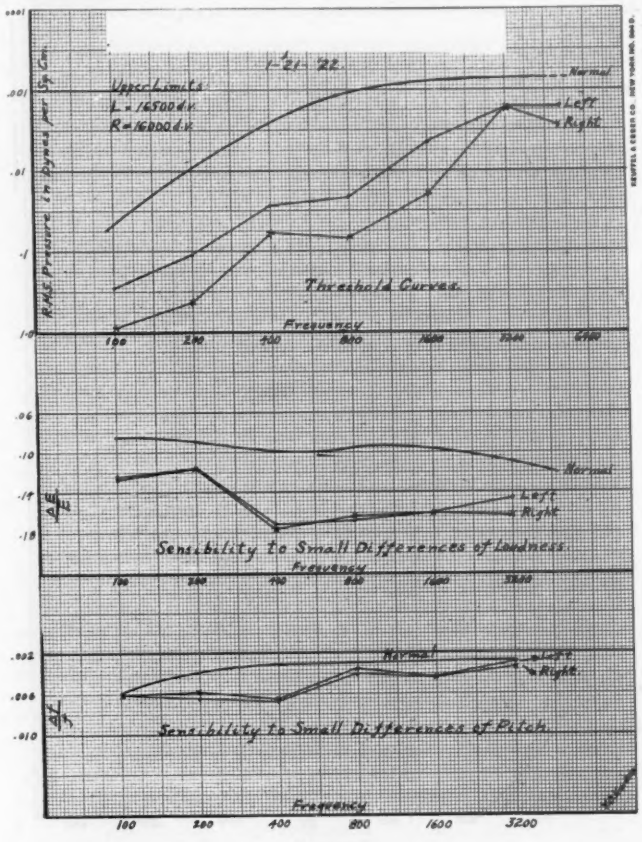


Figure E

The circuit is designed in such a way that a motor controlled key periodically changes, by any desired intervals, the resistance R across which the receiver is connected. The tone emitted by the receiver will therefore periodically and abruptly fluctuate from a

1. Sensibility of the Ear to Small Differences of Intensity and Frequency.—V. O. Knudsen Abstract in Physical Review 19, 261 (1922).

tone of one loudness to a tone of greater or lesser loudness. The two tones are of equal duration and alternate at a rate of about 50 per minute.

If the difference of loudness of the two tones is greater than the smallest perceptible difference for the ear under test the two tones will be heard as a flutter tone, otherwise they will be heard as a

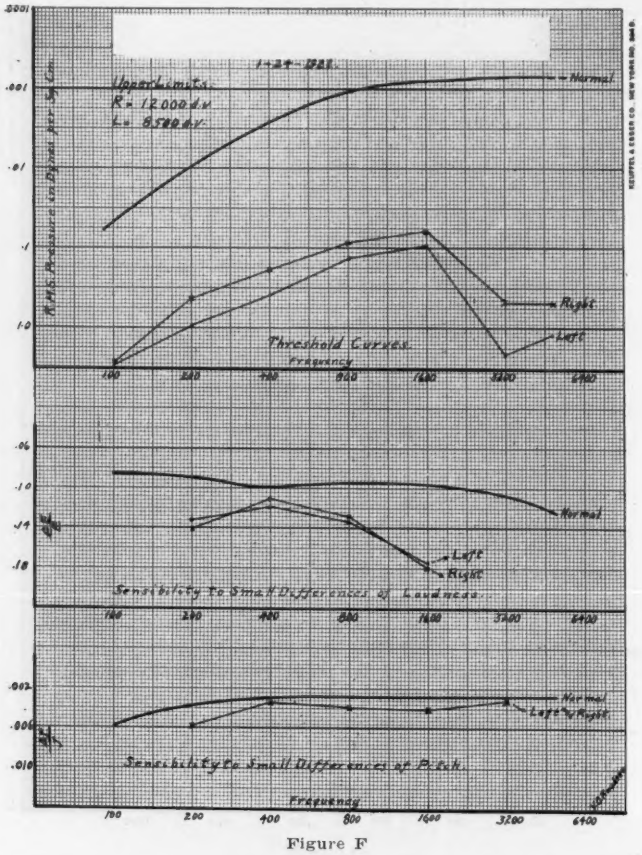


Figure F

steady tone. Hence, to determine for a particular ear the smallest perceptible difference of loudness it is only necessary to properly vary a single slide wire resistance until the person under test decides when the flutter appears as the difference of loudness of the two tones is increased from zero, and when the flutter disappears

as the difference of loudness of the two tones is decreased from a plainly perceptible difference.

A similar procedure is used for determining the smallest perceptible difference of pitch, except that the frequency instead of the intensity of the tone emitted by the receiver is made to alternately fluctuate from a tone of one pitch to a tone of higher or lower pitch.

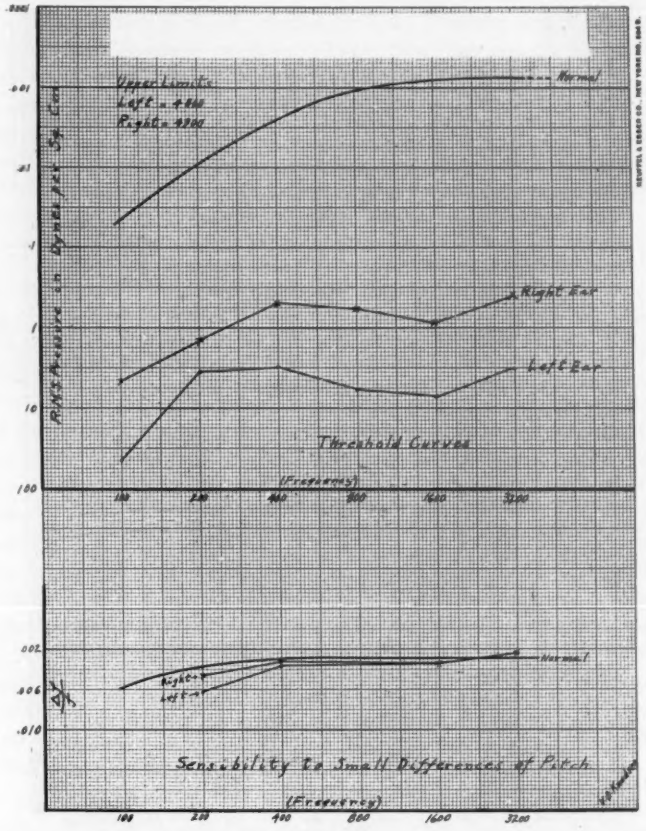


Figure G

Figures A, B and C will show some recent sensibility curves of normal ears. The first slide shows how the sensibility of the ear to small differences of loudness depends upon the intensity of musical tones. $\triangle E/E$ is the ratio of the smallest perceptible difference of energy of a tone to the total energy of that tone. The curves

show that the normal ear can perceive smaller percentage changes of loudness for moderate and loud tones than it can for feeble tones. For moderate and loud tones the normal ear can perceive a difference in the energy of a tone of about 10 per cent. This means that a normal ear can distinguish about 400 gradations of loudness for tones of medium pitch.

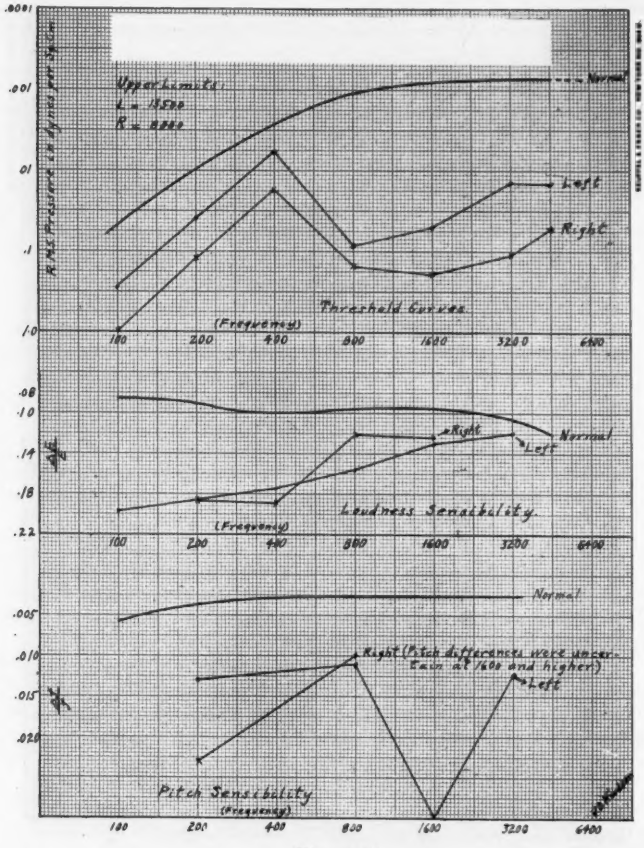


Figure H

Figure B shows how the sensibility to small differences of loudness depends upon the pitch of musical tones. The individual curves of 19 ears and the average curve of the 19 ears are shown. There are noticeable individual differences, but the average curve indicates that the sensibility to differences of loudness is

almost independent of the pitch over the range used in speech and music.

The third slide shows how the resolving power, *i. e.*, the sensibility to small differences of pitch, varies with the pitch of musical tones. $\triangle N/N$ is the ratio of the smallest perceptible difference of the frequency of a tone to the whole frequency of the tone. These curves are similar to those obtained by C. E. Seashore and his assistants at the University of Iowa. They show that the ear can perceive smaller musical intervals for moderate and high tones than it can for the graver tones. The average curve shows that at the higher tones the normal ear can perceive a difference of pitch corresponding to about 1/20 of a semitone. The normal ear can therefore distinguish about 2,000 gradations of pitch within the audible range (20 d. v. to 20,000 d. v.).

The sensibility characteristics of a number of pathological ears recently examined are indicated by the following figures. Each figure consists of curves which quantitatively compare the response characteristics of the ear under test with those of the normal ear. The upper curves compare the threshold intensities of the affected and normal ears. They show simply how much louder the tone must be for the affected ear than for the normal ear. The ordinates give the root mean square pressure in dynes per square centimeter in the external ear cavity. This pressure is proportional to the amplitude of vibration of the tonal stimulus. Thus to produce a barely audible tone for A's left ear at 100 d. v. it was necessary to have the amplitude of vibration about 16 times as great as the amount required for a normal ear. The middle curves compare the sensibility to small differences of loudness of the affected and normal ears. $\triangle E/E$ is the ratio of the smallest perceptible increment of energy of a tone to the total energy of the tone. The lower curves compare the sensibility to small differences of pitch of the affected and normal ears. $\triangle F/F$ is the ratio of the smallest perceptible difference of frequency of a tone to the frequency of the tone.

The curves for the cases presented in the above figures represent the response characteristics of ears affected by varied types of fixation deafness, or intensive nerve degeneration, or diplacusis, or combinations of these. All of the curves indicate that these pathological processes do not greatly affect the pitch and intensity differentiating mechanisms of the ear. In spite of the advanced nerve degeneration in cases "F" and "G" the pitch and intensity differentiating capacities were almost normal. Only one case has been observed

in which the pitch discriminating power was noticeably sub-normal, and in that case it was not serious.

Interesting data on diplacusis are accumulating. Thus far seven cases have been carefully examined. In some cases the affected ear hears too low, in others too high. In some cases the effect extends over the entire audible range of pitch; in others, it is restricted to only a portion of the tonal range. The displacement of pitch identification in the seven cases examined varies from ¼ tone interval in some instances to slightly less than an octave in one other instance. In the case of B, the right ear could not associate the property of pitch with tones whose frequencies were below 600 d. v., yet his response curves indicate that the affected ear could perceive differences of pitch and loudness with nearly the same facility as a normal ear. He said, "A change of pitch seemed like a change in the quality of a sort of a wind sensation in the right ear."

In a subsequent paper the writers hope to give a more complete report of their study of diplacusis, together with the bearing of their data on theories of audition.

People's Gas Bldg.

A CASE OF TELANGIECTASIS OF THE MUCOUS MEMBRANES OF THE NOSE AND LIPS ASSOCIATED WITH LONG-STANDING AND SEVERE EPISTAXIS.*

DR. JOSEPH W. MILLER, New York.

S. W., a widow, was born in Poland, 40 years ago. Her family history, after an exhaustive investigation, is absolutely negative and throws no light on the patient's peculiar affection. As she is an intelligent patient, I was very fortunate to get a definite family and personal history.

The patient, thirteen years in the United States, has two children living and well, one died of pneumonia at the age of two. Had no miscarriages or still-births. She had small-pox at twelve and does not remember having had any other diseases.

Her present condition dates back about twenty-five years ago when she began to have nose bleeds, at first only occasionally with moderate bleeding, and as time went on, the epistaxis became more profuse and frequent; but would come on most frequently while *bending down*. Yet, it would also appear during sleep and while sitting quietly. Only rarely would a week or two pass by without a nasal hemorrhage. But the longer the interval of freedom from bleeding the greater would the next epistaxis be. During all this time the patient would go from hospital to hospital, from dispensary to dispensary, from one specialist to another. She has been admitted and treated in various hospitals on various occasions. Has been given by mouth and by hypo all sorts of drugs calculated to thicken the fluidity of the blood in the hope of checking the nasal bleeding. In addition, the patient has been subjected to countless cauterizations, both by the tyro and the expert specialist. However, all their efforts were in vain, all their methods useless. The patient went from bad to worse as time went on and gradually lost weight and strength. Her anemia resembled the pernicious type and the patient's appearance became frightful.

As she was a frequent visitor to the out-patient department of the Beth Israel Hospital, I had an excellent opportunity to study the patient carefully. I remembered her being in the surgical ward some years ago for the very same condition and carefully checked up all the previous methods of treatment. After several examinations of

*Read before the Beth Israel Hospital Alumni Assn., March 30, 1922.

her nasal chambers and oral cavity I observed very fine dilated blood vessels over both surfaces of the septum and over the mucous membrane of the lips. In fact, on further questioning her, she admitted having occasional bleeding from the right side of the lower lip on the slightest friction. On that part a well marked dilated arteriole is present. On the left side of the septum anteriorly about an eighth of an inch beyond the muco-cutaneous junction is a small mass of granulation tissue the size of a pea, traversed by numerous dilated blood vessels, the seat of the repeated hemorrhages. On the slightest touch of this little tumor, a profuse hemorrhage results. On probing this little mass I found the probe to go upwards and inwards for about an inch into the septum and from the direction of the probe it seemed as if it went through a perforation in the cartilaginous septum to the opposite mucous membrane.

Her face has a death pallor, is markedly pitted, especially over the tip of the nose, as the result of small-pox and at very close range numerous fine pink dilated blood vessels are present all over the pitted area, but are especially marked over the mucous surfaces of the lips and septum. The left nasal chamber is very narrow, due to a marked deviation of the septum on that side. The little mass of granulation tissue on that side of the septum helped to add to the narrowness of the cavity.

On February 1, 1921, a red count was made giving approximately two million red cells, hemogloblin thirty per cent (Talquist). There were few poikilocytes and a good many micro and macrocytes. Serum diagnosis for lues has been repeatedly negative. Two days later she came to the clinic in a frightful condition. Blood streaming from both nasal chambers, which were stuffed with absorbent cotton. Her eyes were glassy and shiny and her face yellowish white. The cotton was removed and gauze soaked in thromboplastin was tightly packed in the left nasal chamber and the bleeding temporarily controlled. Her pulse rate was regular, very feeble and numbered 130 per minute. She was so anemic and weak that I advised admission to the hospital, but the patient refused. With her daughter's assistance she managed to get home, which is only two blocks away from the dispensary. I was called to see her the next morning and found her in bed. I removed the packing and examined the nasal chambers. The little angiomatous mass on the left side of the septum was somewhat larger and slightly bleeding. I made up my mind now to remove this little mass in toto. When I informed the patient of my intention she refused, saying that she is afraid she will bleed to death. From that time on, whenever she came to the

clinic I always advised operation. At one time I even told her that she had nothing to lose, as she would bleed to death anyhow. Finally she took my advice and on March 3, 1921, appeared for operation.

Her nasal chambers were anesthetized as for a regular submucous resection, using pledgets of cotton soaked in ten per cent of cocain solution with a few drops of adrenalin chloride. An incision from above downwards was made slightly behind the muco-cutaneous junction and just anterior to the bleeding tumor mass. As the muco-perichondrium was lifted from the cartilage, I found an oval perforation in the cartilaginous septum just obliquely above the opposing granuloma. Inserting the probe now into the little tumor I found it going straight through the perforation in the cartilage to the mucous membrane on the other side. Instead of removing this inflammatory mass only as I originally planned, I now, in addition, proceeded to do a sub-mucous resection. I removed as much of the cartilaginous and bony septum as was visible to the eye, approximated the opposing mucous surfaces rather tightly by means of Simpson's intra-nasal tampons and ordered the patient to bed. Ice cold compresses to the nose for the next forty-eight hours was the only immediate treatment advised.

I was indeed very much surprised to note that throughout the operation the patient bled almost nothing at all. Of course, the perforation in the quadrangular cartilage can only be ascribed to the frequent use of the electric cautery. That the epistaxis increased after each cauterization is very evident, as it had exposed the opposing mucous membrane through the cartilage.

Forty-eight hours later, I removed the nasal tampons and was rather surprised to find very little bleeding, even less than what usually follows the removal of tampons after submucous resections. She was then seen every other day for two weeks and then told to come once a week. The patient gradually gained in strength, the pulse slowed down to ninety and the face assumed a more healthy color. Since the operation there was hardly any bleeding and only on removing of dried-up secretions with a cotton swab from the operative area did a little oozing appear. On April 21, 1921, a final examination of her nasal chambers was made. The septum was in the mediam line, of a healthy pinkish color and revealed quite a considerable scar immediately behind the muco-cutaneous junction. The red blood count was three million five hundred thousand, hemoglobin sixty per cent and no abnormal erythrocytes. On that day the patient was discharged as improved and advised to call in a few months later.

As mentioned above, all the bleeding came from the little angiomatous mass which was entirely removed during the operation. This was sent to the pathological laboratory and reported back as chronic inflammation.

The most important points in this case are: That one or more dilated vessels ruptured and gave rise to frequent and profuse epistaxis. That due to frequent cauterization of the bleeding points a mass of granulation tissue formed interspersed with blood vessels, which gave rise to still greater bleeding. That due to the use of the actual cautery a perforation was made in the cartilage exposing the mucous membrane of the other side, which gave rise to additional hemorrhage. That due to the removal of the small mass of granulation tissue with resection of the cartilaginous and bony septum, a matting together of the opposing surfaces took place, doing away, for the time being at least, with the frequent and profuse epistaxis. That the dilated vessels present on the septum is but part of a generalized telangiectasis of the face, nose and lips.

But one of the most striking features in this case is the absence of a similar case in the family, nor in two preceding generations. And it is for that reason that the case is here reported.

In the March issue of the *New York Medical Journal*, W. Freudenthal describes a case of "Telangiestasis of the face and mucous membrances of the nose and throat associated with severe epistaxis," in which he claims the presence of an hereditary tendency as absolutely established. He goes on mentioning the observations of Babbington, Legg, Chiari and an exhaustive article by Walter R. Steiner on Hereditary Hemorrhagic Telangiectasia to prove his contention. As my case has no hereditary factor, I began looking for other causes and stopped at that part of her personal history where it says small-pox at twelve.

In looking over the pathological anatomy of this disease I find that in the severe forms the cutis vera is invariably involved. The suppurative inflammation involving its deeper layer—pars reticularis—and as a consequence scarring results. This deep layer of the true skin is traversed by numerous blood vessels, both arterioles and venules which during active inflammation become very much dilated and may never regain their normal calibre. In addition, the scar tissue, ever present as a result of suppuration of the cutis vera, may constrict the blood vessels at one part or another, thus giving rise to still greater dilatations or pouchings of the vessel walls and finally producing telangiectasis.

In the above case numerous telangiectatic vessels are seen crossing the bottom of the pits, but more so over the mucous membrane of

the lips and nasal septum, both parts occasionally involved in small-pox. It stands to reason that this case of telangiectasis of the lips and nasal septum may derive its etiology from her severe attack of small-pox. Moreover, her bleeding began about three years after the attack of variolea, when due to friction of one kind or another one or more of the dilated vessels ruptured, resulting in her long standing attacks of epistaxis.

As I have not found another similar case in the literature, I decided to report this case and perhaps throw a ray of light on the subject from a different angle. Of course, I do not refute the hereditary tendency of this unique affection in a great many cases; but I wish to point out that other etiologic factors may occasionally be responsible for this affection, independent of an established hereditary history. Such causes may be looked for in purely local conditions of an involved area, as *e. g.*, small-pox, erysipelas, cellulitis or phlegmon and deep seated burns. Extensive and deep suppurations with consequent cicatricial changes in a given locality may in addition to other deformities give rise to telangiectasis.

268 E. Broadway.

A NEW ARTERY CLAMP FOR TYING OFF DEEP TONSILLAR VESSELS.

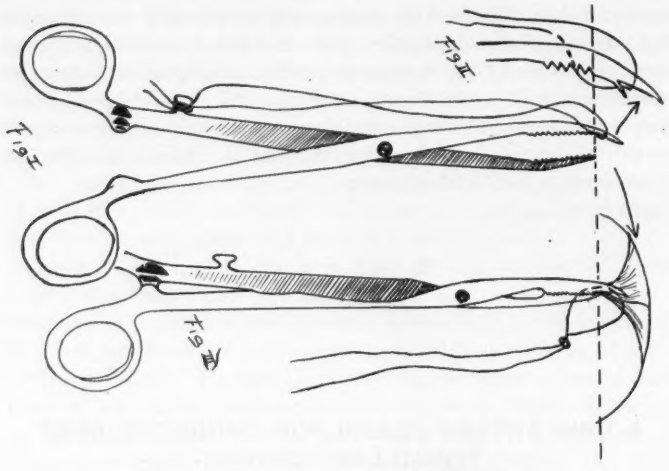
Dr. Alfred Kahn, New York.

The usual procedure in tying off tonsillar bleeders, following the total dissection of the tonsils, is as follows: The bleeding vessel is grasped by a pair of long artery forceps. A ligature is then tied loosely around the clamp, slid down the clamp, over its nose and then around the artery, where it is firmly tied. The whole procedure is simple, except the "getting" of the ligature around the nose or apex of the artery clamp. The ligature often has a tendency to tie down on the nose of the instrument—it does not slip over.

The instrument shown herewith was made with the object in mind of overcoming the difficulty mentioned. Fig. 1 illustrates the instrument. It differs from the ordinary artery clamp in that one blade is slightly longer than the other. The overlength is at the tip, and prolongs the blade as a sharp needle-like extension. This extended

tip is notched on its inner edge in such a way that a ligature will notch into it. Fig. 2 is an exaggeration, showing detail of tip.

Technique. The point of bleeding is noted. The opened forceps reach down to this spot. Before grasping the bleeding point, the pointed blade is pushed slightly under the bleeding vessel. This movement carries the ligature that is notched on the blade into the tissue beyond and below the bleeding point. The clamp is closed and the vessel grasped. The long ligature, both ends of which have been carried back and wound around a button projection from the other blade at its finger end, and that had been mounted on the



clamp before the clamp was inserted into the mouth, is now loosened from this button. A knot is made and carried down beyond the tip of the artery clamp, where it is tied. This movement is shown in Fig. 3. The ligature is carried into the tissue distal to the point of the clamp. The ligature is always beyond the clamping part of the forceps, so that when it is tied it cannot possibly touch the end of the clamp. The bleeding point having once been grasped, the tying of the knot is simple.

48 East Forty-ninth Street.

CAUSES OF FAILURE IN SURGERY OF THE NASAL ACCESSORY SINUSES.

DR. WILLIAM MITHOEFER, Cincinnati.

During the past fifteen years, intranasal operative measures for the relief of accessory sinus disease have been the methods of choice by the majority of rhinologists. We have been able through our mechanical skill and our knowledge of the anatomy, to deal radically with the affected cavities, but, unfortunately, even after a most thorough intranasal removal of all diseased tissue, we have often asked ourselves the following questions: Has the end justified the means? Is not the patient in the same or worse condition than before the operation? Why have we failed in our endeavor? If we attempt to analyze carefully the various factors that play a role in the causation of our failure, we find that in many instances a probable explanation may be given.

The most important causes of failure are:

1. Lack of knowledge regarding the anatomic variations of the accessory cavities, especially of the ethmoid.
2. Negligence in not making a general physical examination of the patient before operation.
3. Lack of persistence in trying to relieve the patient with conservative treatment of the nose.
4. Insufficient consideration and examination of all sinuses and the nasal mucosa before operating.
5. Lack of knowledge regarding the character of the infection.
6. General anesthesia.
7. Insufficient after treatment.

Anatomic Variations. This phase of the subject deals principally with the lateral displacements of the ethmoid labyrinth, for the detection of which a careful study of the skiagram is necessary. It behooves us to give a guarded prognosis as to the ultimate success of an intranasal operation when the skiagram shows the presence of an orbital extension of the ethmoid labyrinth. It is in these patients with laterally displaced ethmoid cells that failure most often occurs. Chronic disease of the orbital cells, whether suppurative or hyperplastic, gives rise to symptoms similar to those of frontal sinus involvement, and it has been our experience on several occasions in doing a radical frontal sinus operation to find the frontal sinus absolutely healthy, but the orbital ethmoid cells very much involved.

We have been impressed in these cases by the futility of endonasal surgery, and in some cases firmly believe that the external ethmoid operation should be the method of choice, and that endonasal measures should not be attempted. Primary radical ethmoid surgery is indicated in only well selected, carefully studied cases, and in all patients showing evidence of meningeal involvement. In the majority of cases, on the other hand, it is advisable to begin with an endonasal operation, for it may happen that drainage of the orbital cells or the widening of the nasofrontal duct will be sufficient to relieve the patient of an obstinate headache.

Anatomic variations of the frontal sinuses are of frequent occurrence. When the skiagram shows the frontal sinus opaque and divided into numerous compartments, we may expect little or no benefit from an intranasal operation. The same is true when the interfrontal septum of the healthy side is displaced to such an extent, that it causes a narrowing of the nasofrontal duct leading to the affected frontal sinus of the opposite side. A large frontal sinus with a well marked temporal recess is another anatomic variation that may be the cause of our failure. A mucocele or pyocele of the anterior ethmoid cells, extending upward into the cavity of the frontal, is a condition met with occasionally in doing a radical operation. In one case which was operated upon during the past year, the pyocele was as large as a pigeon's egg, so that there was complete closure of the frontal ostium. The only part of the frontal sinus not occupied by the pyocele was a small temporal portion which was found full of granulations and inspissated pus. It is obvious that intranasal surgery is of no avail in cases with this kind of anatomic variation.

The width and position of the nasofrontal duct is another interesting anatomic study. The duct is narrowed by the presence of enlarged subfrontal infundibular cells with or without intrafrontal dilatations. When the subfrontal cells are enlarged, they may also displace the nasofrontal duct in various directions. It may be displaced anteriorly, posteriorly, to the midline, or towards the orbit in a markedly lateral direction. A high bony septal deviation may also be the means of closing the duct.

In considering the anatomic factors pertaining to the maxillary sinus, we must mention the various recesses such as the malar, alveolar, palatine and prelacrimal, that are often found well developed, and cannot be reached except by radical means. We must also remember that bony septa occasionally divide the cavity into several compartments and, furthermore, that the floor of the max-

illary sinus, especially in the adult male, lies at a much lower level than the floor of the nose, so that no matter how radical the operation, infective processes may continue to exist in this region.

So much for the anatomic phase of the subject. Summing up what has been said, it becomes manifest that the anatomic variations play a most important part in preventing us from getting good results in our surgery of the nasal accessory sinuses. If we remember these various anatomic reasons for failure, we shall be able to treat more intelligently some of the intractable cases that come under our observation.

It may not be amiss in this connection to say a few words concerning the skiagram. It is our opinion that a skiagram is dispensable for the making of a diagnosis of accessory sinus disease, but that it is indispensable if an operation is contemplated on either the ethmoid or the frontal sinus. Every well trained rhinologist should be able to make a diagnosis of accessory sinus disease by means of the various diagnostic methods at his command, and he should rely on the skiagram only as a means of informing himself regarding the anatomic variations of the cells. We consider the X-ray picture as an adjunct of secondary importance. The reason for this is quite obvious. We have seen skiagrams with marked opacity of the various sinuses, in which there was no clinical evidence of disease. We have seen others where clinically there was present every evidence of involvement, but the Roentgen picture was negative, so that he who jumps at definite conclusions from the appearance of the skiagram alone is making a grave error.

The second cause of failure to be considered is our *negligence in not making a careful physical examination of the patient prior to operation.* Advanced age, for instance, is certainly a plea for conservatism, and an operation should not be attempted unless the symptoms are of such severity as to make the interference absolutely imperative. In operating on older patients, great care and gentleness must be exercised on account of the brittleness of the bones and the danger of fracturing the cribriform plate.

The most important factors to be considered in the general examination are the condition of the blood, urine, sputum, eyes, teeth, tonsils, heart, lungs, intestinal tract, ductless glands, and the muscles of the neck. A careful analytic study of each patient will often reveal some disturbance of the general state of health, and consequently make us more conservative. Take, for example, a patient in whom there is present a mild hyperplastic ethmoiditis with severe headache. Examination of the eyes shows the presence of a mus-

cular asthenopia which after proper correction entirely relieves the headache. It is true the patient still has a chronic ethmoiditis, but it is of little consequence since the headache, which was the chief complaint, is relieved and the symptoms of the ethmoiditis are mild in character.

There is another type of patient in whom the nasal mucosa is constantly in a state of chronic infiltration, and who seeks medical aid for the relief of an incessant headache. A careful study of this patient shows that there are present symptoms of hypothyroidism. The insufficiency of the thyroid is usually of the benign type without any external evidence of myxedema. The cellular protoplasm of these patients is constantly overloaded with mucin and fat, and the headache is probably due to an infiltration of nerve cells and of the meninges. We have observed cases of this type in which headaches disappeared after the use of small doses of thyroid extract over a long period. Some of these patients state that, while taking the thyroid, the insomnia from which they had long suffered disappeared, and that the constipation was overcome. The intestinal stasis, which is a characteristic symptom of hypothyroidism, is probably due to a muscular infiltration of the intestinal wall. The study of the endocrines is still in its infancy and gives much food for thought.

It is a characteristic fact that the removal of the tonsils will often relieve a headache of long standing, and occasionally do away with an intractable case of vasomotor rhinitis. The extraction of a tooth may clear up an old antrum infection and an impacted third molar may be easily overlooked.

Very few rhinologists, in making a routine examination ever consider the condition of the muscles of the neck. There is often present a myalgia (probably the result of some focus of infection) which causes severe headache radiating from the occiput to the forehead. The muscles of the neck are hypertonic; the sternoclavicular joints and the cervical vertebra are painful on pressure. A marked case of myalgia of the neck present in a patient with accessory sinus disease will seldom be relieved after operation. Other means, such as mild massage, injections of calcium chlorid, sodium chlorid and novocain solutions into the muscles may overcome the hypertonicity and relieve the headache. There is much to be said regarding the examination of the muscles of the neck, but time will not permit me to touch upon the subject except in a cursory way. It behooves us, however, to give these muscles the attention they deserve.

Chronic bronchitis with bronchiectasis is often present in suppurative accessory sinus disease, and should be recognized before opera-

tion. In these patients, the cough does not always disappear unless other therapeutic means are used, even though the accessory sinus suppuration has been eradicated.

It is needless to say that a Wassermann test should be made on every patient upon whom a sinus operation is contemplated. We have made a Wassermann on all cases, and have been impressed with the fact that syphilitic involvement of the accessory sinuses is present many times when least suspected.

Let us now consider the third cause of failure—*lack of persistence in trying to relieve the patient with conservative treatment of the nose.* There are some patients who complain of many of the symptoms of nasal sinus disease, in whom we cannot clinically demonstrate the presence of a sinus involvement. These patients usually suffer from a form of nasal neurosis and are rarely benefited by surgery. It is true that a great many of them have a latent ethmoiditis, usually of the hyperplastic type, but in the majority of instances, even after a most thorough and painstaking operation, the patient continues to sneeze, has the same hypersecretion and headache as before. To prevent failure in these cases, the nasal neurosis must be the primary consideration and be properly treated, even though there may be clinical evidence of a mild ethmoiditis.

Conservatism should be the first consideration in all mild types of inflammation of the accessory sinuses. Simple means such as the correction of a nasal obstruction, the infraction of the middle turbinate, the application of various astringents to the region of the spheno-palatine branches, and the anterior ethmoid nerves will often relieve patients of their distressing headache and render an operation unnecessary. Good results after radical procedures in these cases of mild ethmoiditis are less frequent than after conservative treatment.

We now come to a very important part of this subject and one that is very often the cause of failure, namely, *insufficient consideration and examination of all the sinuses and the nasal mucosa before operating.* The method of procedure in the operation will have to depend entirely on the clinical findings and the results obtained from the use of the various diagnostic measures at our command. Take, for instance, a maxillary sinus suppuration. The first requisite in these cases, after the teeth as a causative factor have been excluded, is to determine whether the antrum is primarily involved, or whether it is only acting as a reservoir for pus from above. It very often happens that the inferior hiatus cells are diseased, and are the sole cause of the antrum suppuration. Unless these cells

are dealt with at the time of operation on the antrum, our endeavor will be met with failure. The antrum continues to discharge pus, and the patient may be in a worse condition than before the operation. Especially is this true if a part of the inferior turbinate has been sacrificed. In these cases, there may be added to the old trouble another distressing complication in the form of a post-operative rhinitis sicca with crust formation. It is therefore far better in many of these cases to approach the antrum from above by removing the hiatus cells and enlarging the natural opening of the antrum. This is a conservative operative procedure and gives better results than an opening made in the inferior meatal wall. When pathologic changes have taken place in the antrum, a more radical procedure must be adopted, but, in most cases, the hiatus cells must be thoroughly removed.

Marked involvement of the ethmoid labyrinth may be present without affecting the frontal sinus, but it rarely happens that the ethmoid labyrinth escapes infection if there is present a chronic frontal sinus disease. If sufficient attention is not given the sphenoid cavity in the course of the examination, pathologic changes present in this region may easily be overlooked. I doubt very much if suppuration of the sphenoid cavity ever exists without some involvement of the adjacent posterior ethmoid cells. Simple hyperplasia of the anterior sphenoid wall, with consequent closure of the ostium, is often sufficient to cause severe symptoms. The lining membrane of the sphenoid cavity in many of these cases is not hyperplastic. The various eye and nerve manifestations that occur are probably accounted for by the circulatory changes that take place from closure of the ostium. In patients with recurrent polypi, it becomes necessary, if a good result is to be obtained, to examine carefully the condition of the antrum. If the skiagram is opaque, far better results are obtained if we begin the operation by dealing radically with the antrum and removing all polypi, which are usually found filling this cavity. The ethmoid labyrinth is dealt with at the same time. If, after such radical procedure, polypi still continue to form and make their appearance in the region of the naso-frontal duct, we may be reasonably certain that the orbital ethmoid cells and frontal sinus are also involved. If the symptoms demand an interference, an external ethmofrontal operation will give the best results. We saw recently a patient who had sought medical aid for nine years for relief from a severe nasal hydrorrhea with chronic eczema of the lip and recurrent polypi. He was under our observation for over a year, during which time radical operations were done upon all the sinuses. He was not entirely relieved until after a bilateral external

ethmofrontal operation. Failures in these cases will be less frequent if we begin the operative work in the antrum and radically remove every vestige of polypoid tissue.

In all atrophic states of the nasal mucosa, we must proceed with caution, being as conservative as possible. Operation in the presence of atrophy is often followed by crust formation which may continue indefinitely. If radical surgery is necessary in these cases, care must be taken not to curette the atrophic membrane of the accessory sinus.

It is necessary in ethmoid surgery to operate with more gentleness, and to abstain as much as possible from indiscriminate curetting of the mucous membrane with sharp instruments. If the middle turbinate is not too large, it should be displaced to the middle line as a primary step of the operation. There are various good reasons for the preservation of the middle turbinate in ethmoid surgery. In the first place, we are retaining a functioning mucous membrane, rich in glands. We are preventing the formation in the middle meatus of a large open and raw surface which is often the cause of great discomfort in cold weather, and predisposes the individual to recurrent attacks of infection. We are, furthermore, preventing the possibility of a post-operative rhinitis sicca, and lastly and most important of all, we are making the operation less dangerous by working to the lateral side of the median plate, away from the olfactory sheaths which are in direct communication with the arachnoid space. Post-operative meningitis should be of rare occurrence if the middle turbinate is preserved.

We will next consider the *bacteriologic* side of the question. Failure to make a bacteriologic examination of the nasal discharge before operation will often lead us into difficulty and may endanger the life of the patient. We should certainly not operate in the presence of a streptococcus hemolyticus infection unless urgent symptoms are present, and then only in a radical way, in order to insure perfect drainage and ventilation. Furthermore, we should consider it poor surgery to operate when there is present a large quantity of pus, and would rather treat the case, for the time being at least, in a conservative way. When atrophy with or without ozena is present, we usually find the bacillus mucosus. When these organisms are found, we may depend on the case being an intracable one. The staphylococcus albus usually predominates in all of the chronic suppurative cases. During an acute exacerbation other more virulent organisms prevail and their presence at this time undoubtedly adds to the danger of the operation.

We have mentioned before that we consider the *administration of a general anesthetic* to be one of the causative factors in our failures. Our experience during the past two years with the use of the local anesthesia in all radical accessory sinus surgery, including the radical frontal operation, has convinced us that better and more thorough eradication of all diseased cells can be done with this method. There is little bleeding, the operation is technically easier to perform, the patient has less shock, and the operator is never hurried and can leisurely examine every recess that may if overlooked cause a post-operative retention.

There are other factors, too, that prevent us from getting good results in radical frontal sinus surgery. We cannot hope for a good result if we use the same method of procedure in every case. We must vary our technic with the anatomic situation of the cells.

One of the most important parts of the operation is the removal of the orbital prolongations of the ethmoid labyrinth, so that any operation on the frontal sinus that fails to uncover these cells is not sufficiently radical to prevent post-operative retention. Under local anesthesia, it becomes a simple procedure to approach the sinus from the orbital wall as described by Ritter. By opening the sinus in this location, there is also less danger of meningitis. Post-operative meningitis after frontal sinus operation is often caused by a thrombo-phlebitis of the diploic veins in the anterior wall of the sinus. If we are able to simplify the radical frontal sinus operation by the use of local anesthesia, by treating each case according to the anatomic findings, and by not entering the sinus, unless necessary, through the anterior wall, we shall obtain better results in our work.

A carefully executed operation often fails to give a good result on account of *insufficient after treatment.* This applies chiefly to the ethmoid region. Patients must be treated at regular intervals after the first week, and edematous mucous membrane cauterized until the entire cavity has healed with a smooth scar. Good results seldom take place if the after treatment is neglected.

In conclusion, let me impress upon you again that the proper study of each individual patient before operation is absolutely imperative. If all the known causes for failure have been duly weighed in the balance, our results will be more satisfactory. There has been a tendency during the past few years among medical men to deprecate the value of accessory sinus surgery. This state of affairs is undoubtedly the result of indiscriminate operating without due regard of the many conditions, both local and general, that should always be investigated when an operation is contemplated. If a careful

analytic study is made of each individual patient, better results will be obtained and there will be less danger of surgery of the nasal accessory sinuses falling into disrepute.

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RECORDING OF FUNCTIONAL HEARING TESTS.

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While hearing tests are used quite similarly by different otologists and the significance of these tests generally accepted, the method of recording the results differ so widely that it is almost impossible to compare the findings reported by one observer with those of another, unless both describe the methods used in recording or explaining each reaction.

The recording of visual function has been standardized to great exactness with universally accepted symbols, and while hearing tests offer greater obstacles in exact recording greater uniformity in methods can and should be brought about.

Conduction apparatus defects are characterized by poor hearing for low tones, relatively good hearing for high tones, shortening of air conduction with increase of bone conduction; while perception apparatus defects are characterized by impaired hearing for high tones, relatively good hearing for low tones, and decrease in bone conduction more marked than air conduction. We have then to deal with both quantative and qualitative estimation of hearing function in our tests.

It is quite obvious, therefore, that to approach some measure of uniformity in our tabulation of functional hearing tests some method must be adopted that will give approximately the same results by different observers, and at different times, and be simple and efficient enough to be easily understood by different observers.

It is impossible to make tuning forks, or noise-producing apparati that are exactly uniform, and it becomes necessary to determine the normal for each individual instrument for a standard. Politzer attempted to produce a device for measuring hearing in the Acoumeter, but one has only to examine several such instruments at the same time, to convince himself that even in these there is a difference in intensity of sound.

The charting of hearing results with forks, as is generally done at the present time is a very relative matter, and, while the general result is appreciable to others, the exactness of finer gradations is entirely lost. Such notations as "Bone conduction increased;" "Air conduction decreased;" or "Lowered high tone perception," etc., qualified by "slightly," "moderate," "marked," etc. give a relative appreciation to another observer, and while they are probably sufficient to make a diagnosis in a given case they are not sufficiently accurate to show any but gross changes occurring in the patient's hearing as found at a subsequent examination either by the original, or by another observer.

The estimation of quantitative hearing for a fork precludes a normal hearing for that fork, which is best estimated by measuring the time the vibrations are audible. The method commonly in use of estimating that normal by the observer's own hearing is open to the criticism that the observer's hearing must be normal, or due allowance made for his own defect, which if present still complicates the test by essentially requiring two tests to be recorded, i.e., both the observer's and the patient's.

For other tests, such as voice, watch, and acoumeter which are measured by distance heard from the ear, many factors are present which vary the accuracy of the test. These tests are valuable, but can be only relatively exact, due to acoustics of the room in which the patient is examined, extraneous noises, atmospheric conditions; and in case of voice tests, the present condition of the observer's vocal apparatus.

A GIVEN TUNING FORK SET OFF AT A GIVEN INTENSITY WILL AUDIBLY VIBRATE FOR A GIVEN EAR, FOR A CERTAIN TIME. This axiom forms the basis of our system. The difficulty here is to start the vibrations of the fork with the same intensity each time. Very likely there is no absolutely precise method that is practical for this purpose, but setting off of the fork by striking it on a cork, or pad, striking the fork with a pleximeter, or snapping the tines between the thumb and finger is sufficiently accurate. One can readily satisfy himself that great accuracy can thus be attained. Measuring the audible vibrations of the fork by a stop-watch he will find but a few seconds of vibrations in repeated tests, and surprising uniformity in setting off of the fork. In our experience snapping the fork between the thumb and fore-finger has been the most satisfactory method of initiating the vibrations.

After selecting a favorite method of setting off of the fork, a normal hearing time is established for that vibration intensity by testing many supposedly normal ears—an average is then taken and is recorded as the normal hearing time for that fork. Each observer must be sufficiently familiar with a given set of forks to appreciate their quoted normal time. In the tabulation of hearing for any fork, it is desirable to have its estimated normal hearing time appear in the formula for comparison with that of actual time heard. Tuning forks must be without overtone, and the longer the vibration, the more delicate the test.

At the Eighth Otological Congress at Buda Pesth, the Uniform International Acoumetric Formula suggested by Politzer, and Gradenigo was adopted. It is most ingenious, in that it comprises all the hearing tests in a small space, and can be used with any set of forks, after they have been standardized. The exact hearing for any set of forks can be recorded so as to be clearly appreciated by others as the normal, for that particular set is part of the formula. I do not understand why this formula and system has been so little followed, bearing as it does the stamp of approval of that Congress, and filling the lack of any other uniform system. This system has been followed by myself and office associates for eight years with great satisfaction. We have made a few minor changes to suit our individual needs. Recently we have adopted its use in our County Hospital Clinic, where our staff can further test its value. At

our Clinic, we are all using one set of forks consisting of the Kitching 64 D.V.; The Edelmann, 108.75 D.V. for Schwabach & Weber's Test; the 435 D.V. for Rinne and the 3072 D.V. for high tone test.

Any set of forks can be standardized for this formula so that the individual preference for various forks is not interfered with in the following-out of the system.

A. D.

$$W : S^{(20)} : A^{64Dv}_{(35)} : A^{108\text{-}75}_{(75)} : A^{435}_{(60)} : A^{3072}_{(35)} : M^{435}_{(25)} : R : V : v : G : W$$

A. S.

The formula consists of symbols which are the abbreviations indicating the various tests. These are placed on a horizontal line. Following each abbreviation, is the number of vibrations of that fork, below which, is its estimated normal hearing time in seconds. The length of time a fork is heard is recorded in seconds. Notations for the Right ear are written above the characters; while for the Left ear, they are written below the characters, and can thus be compared to the normal at a glance. The first letter "W," stands for Weber's Test, the lateralization of the tone being indicated by a small dash (-), or arrow, above or below, for right, or left, respectively. If there is no lateralization, an equals mark (=) is placed after the letter. The next letter "S" stands for Schwabach, the normal time being indicated when the fork is applied to the saggital plane of the skull. Notations for this reaction are recorded plus or minus (signs), with the number of seconds varying from normal—e.g. normal time for a fork is twenty seconds, and is actually heard ten seconds. The notation is minus ten(-10). If Schwabach is normal, an equals sign is placed after the symbol. The next letters, "A" indicate Air conduction for the various forks desired to be used in Air conduction. For routine examination only low and high forks are needed. The next letter "M" indicates Bone conduction over the Mastoid. The fork here used by us is 435 D.V., and as this same fork has its air conduction recorded, the Rinne reaction can be at once estimated. The next letter "R" stands for Rinne and is tabulated with plus or minus (signs). The next letter is "V," and stands for conversational voice. The next smaller "v," stands for whispered voice, and can be marked in fractions—as twenty foot voice, e.g. 3/20 written

above indicates twenty foot whispering heard at three feet in A. D.

If the Llarm Noise Apparatus is used a large "L" is drawn over the "V;" thus indicating the hearing has been taken with the other ear Blocked. This test is not usually necessary except in cases of gross hearing defects, or certain Medico-legal cases.

The next letter. "G" stands for Gelle, and is marked plus, or minus (signs)—as to whether positive or negative. The next character is "Wh" indicating whistle (Galton, or Edelmann). This is the formula we use in our routine work. We usually fill out only part of the tests in a given case, without wasting space on the history Chart as the stamp is so small. Other symbols may be used to supply individual habits of testing; thus, L. I. Limes Inferior, or Lower tone limit; L. S. Limes Superior, or high tone limit. "H," or Horologium, for those who use the watch test, after which space may be left for normal distance of a certain watch.

The whole formula is supplied by a rubber stamp, requiring but a small space on the Chart and does not materially interfere with other notes as do many of the large formulas used, some of which cover nearly a whole page of the Card. The stamp being small, it is more easily transferred than the square type of stamp to the history Card. This is particularly appreciated in Clinics, where economy of space on history Chart is desired. It is very convenient to use so small a stamp on the history Chart, or Card, in the space for notes of that day, and being distinctive in appearance calls attention to the days on which hearing has been tested in looking back over history.

Tests, after inflating the ears, can be noted above and below the numbers that were found before inflation, for the right and left ears respectively thus any changes brought about by the inflation noted at a glance.

No originality is claimed in this paper for this formula; it is presented fully in Barnhill & Wales's Text Book, "Modern Otology," 1911 Edition. After extensive use of this System, we most heartily endorse it and make a plea for its more general use by Otologists in this Country.

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DEATH FOLLOWING OPERATION FOR REMOVAL OF TONSILS.*

DR. FRED W. BAILEY, Cedar Rapids, Iowa.

A few months ago, in preparing a paper on "Control of Hemorrhage in the Tonsil Operation," I sent out a questionnaire in which one of the questions was, "Have you ever had a death from hemorrhage following operation for removal of the tonsils?" I received three hundred and fifty answers to this question, and was greatly surprised to find that, out of every thirteen operators who answered the question, one had had a death from hemorrhage following operations of this sort. The doctors who answered this question reported from one thousand to twenty thousand operations each. I was therefore unable to compute the exact percentage of deaths from hemorrhage following tonsil operations, but the fact that one in thirteen operators had had a death, caused me to think that it might be of importance and instructive to write a paper on deaths following tonsil operations, no matter whether by hemorrhage or otherwise.

I myself have never had a death from hemorrhage following an operation for the removal of tonsils, but in a series of about four thousand cases, I have had two deaths which might be indirectly attributed to removal of the tonsils. These cases I shall report later on in this paper.

By careful search of the literature in some of the leading libraries of the country, back as far as 1910, I find the following cases reported:

Dr. I. G. Clark in *The Ohio State Journal of Medicine*, September, 1921, Vol. XVII, page 9, in an article entitled "Fatalities Following Operations Upon the Tonsils," states that in his estimation the death rate from such operations are of the ratio of one to two thousand. He also states that other writers consider that one to eight hundred percentage would be more fair. In the same article he cites nine cases which were gathered from the vicinity of Columbus, Ohio, which resulted in death. Of these, one case was from septicemia after five days, one from cocain poisoning at the time of the operation, four from pneumonia, and one dying on the table at the time of the operation without regaining consciousness. He also reports that a certain large insurance company states that in 1920

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in 3,944 death claims reported, four had died following tonsillectomies, the ages of those dying being twenty-three, thirty-three, forty-five and fifty-eight years. Two were from septicemia, one from pneumonia, and one in the course of operation. Another insurance company reported 25,927 deaths, of which eight had died from tonsil operation. Seven of these died the same day and one died nineteen days later.

The Bureau of Vital Statistics of Ohio in 1920 reports fourteen deaths following tonsillectomy.

In the *Journal of Laryngology and Otology* for April, 1921, Vol. XXXVI, page 4, Dr. T. B. Layton of Gray's Hospital, London, in a paper entitled "Deaths After Tonsillectomy," reports one case. This case he operated upon with local anesthetic, novocaine and adrenalin, but, as the patient bled considerably, he decided to give a general anesthetic and the patient was put to sleep with ether. The patient bled a great deal at the time of the operation; so much so that the tonsillar fossa had to be sutured. The patient recovered from the anesthetic, but after a few hours bled a great deal more and finally died in collapse about twelve hours after the operation.

In the *American Journal of Medical Science*, 1910, Vol. 140, page 399, Dr. F. R. Packard reports a case of a child three and one-half years old, who died following an operation for removal of the tonsils. The child was operated upon under a general anesthetic, the anesthesia being commenced with Ethylchloride and continued with ether. This operation was started about ten in the morning, at which time the patient bled considerable. At about 5:00 p. m., the patient suddenly had a choking spell and died while the interne was trying to clear the throat with the aid of a tongue depressor and sponges. Tracheotomy was performed at once and the trachea was found to be free from blood clots or foreign matter.

In the *Medical Record*, Vol. 93, page 19, June 29, 1918, Dr. William W. Carter reports a death following tonsil operation in a girl five years old. The patient was given ether about 2:55 p. m. A Sluder operation was performed and the patient died at 9:55 p. m., after a sudden attack of cyanosis. Post mortem showed no post-operative hemorrhage, but a very large thymus gland, and diagnosis of status lymphaticus was made.

In the *British Medical Journal*, Vol. II, 1920, September 18, Mr. Alban Evans reports a case of death following operation for removal of tonsils about half an hour following, due to collapse. Post mortem showed a very large thymus and thyroid gland. A diagnosis of status lymphaticus was also made in this case.

It certainly seems to be true, as many writers have stated, that the tonsil operation has been and still is taken entirely too lightly. Patients come to the office, are told that they should have their tonsils removed. They consider that the matter is a very trivial one, that they only have to go to the hospital or come to the office and have the work done and expect to be ready to go to work in a day or so. As a rule, the element of danger never seems to enter into the operation at all. The time has come when this general idea should be overruled and the patient should be told outright that when he has his tonsils removed he is undertaking an operation which is fully as dangerous as having his appendix removed, and as a rule causes him a great deal more discomfort and pain. The danger of death following this operation should not be underestimated. The laity always attribute a death to the last operation performed, providing it has occurred within a reasonable time after the operation. As a general rule, I would say that if the patient has his tonsils removed and dies within fourteen days, this pateint's friends and relatives as well as the general public will say that the operation killed the patient, and perhaps they are correct. It seems to me that cases of death following this operation are not reported as often as they should be. There is no reason and no excuse why every death following such an operation should not be recorded. I know personally of six cases of death following this operation which have occurred in my immediate vicinity, none of which have been reported.

I now wish to report my own two cases of death which have followed operation.

Myrtle Shaffer, aged five, referred to me by Dr. Skinner, of Marion, Iowa. This little patient was brought into the hospital on the morning of March 4, 1920. On that morning, I had eleven other tonsil cases. Dr. Skinner brought the little girl in and asked me to remove her tonsils, because she had had a great deal of throat trouble. Her tonsils were enlarged and she also had symptoms of adenoids. The usual examination was made in the child's room; that is, the urine was negative, as was also the blood test. The child was brought to the operating room and anesthetized with ether. When I started to remove the child's tonsils, I noticed that they were very abnormal, both in size and in appearance. I made the diagnosis at the time of hyperkeratosis of the tonsils and called the attention of the internes and several visiting doctors to the condition. The tonsils were removed by the Sluder method and the adenoids removed with a La Force adenatome. At the time of the operation, the patient did not lose as much blood as one would expect in any case,

and was sent from the operating room in good condition. I started on the next case, when one of the nurses came to the operating room and stated that the little Shaffer girl was bleeding more than she should. I told them to bring the youngster back to the operating room and on examination, it proved that she was hemorrhaging more than customary. I had the interne administer ether and as soon as I finished the operation in which I was engaged, I placed two or three cat gut sutures around the bleeding points, clamped off the adenoid vessels and thus controlled the hemorrhage. However, the hemorrhage proved to be quite troublesome and it was perhaps thirty minutes before completely controlled.

The little patient was then sent to her room and she seemed and appeared to be as any ordinary tonsil case. I saw the youngster at 1:00 p. m., at which time she had no hemorrhage, appeared to be perfectly all right and had water to drink and was comfortable.

About 4:00 p. m., the interne, on making his rounds, saw the patient, at which time she complained of being hungry, was sitting up in bed and apparently in good condition. Fifteen minutes later the little patient suddenly became cyanosed, and by the time the interne got to the room after being called, the patient was apparently dead. Artificial respiration and oxygen were administered without any avail. The interne stated that the patient apparently died of respiratory failure, as the heart seemed to beat for some time after respiration ceased. An autopsy was not obtained.

My opinion is that the patient died of embolism of the respiratory center. Death in this case was probably due directly to the operation, and I think there is no question but that the youngster would still be alive if the operation had not been performed.

Mr. Arthur B—, aged 32, married, came to me on September 10, 1920, to have his tonsils removed. I had taken care of this patient twice before, within the last year, for a very severe case of quinsy. The operation was performed with a general anesthetic, gas and ether being used. The operation was successful with no hemorrhage to speak of and the patient made a very fine recovery. He left the hospital in three days and six days after the operation drove his car to Burlington, Iowa, a distance of about eighty miles, where he attended a dance. He returned the following day and went back to work in a general wholesale house and continued to work until the fourteenth day after his operation.

The evening of the fourteenth day, he attended a dinner party and ate two bowls of canned soup. Several of the guests were sick, but he seemed to suffer more than the others, being distressed by much

nausea and vomiting. The next morning, he called his general physician, who in turn called me, stating that the patient probably had odema of the larynx. I went at once to examine the patient, but found the larynx absolutely normal. However, the patient was suddenly unable to swallow. He seemed to feel perfectly well and had a temperature of 101°, but otherwise than being unable to swallow, seemed perfectly normal.

I deemed it best to have the patient taken to the hospital and this was done in the afternoon. His difficulty in swallowing gradually became exaggerated and it was decided to have consultation and Doctor Howard from Iowa City came and examined the patient. He thought perhaps the condition might be one of tetanus and administered tetanus antitoxin.

That night the patient became much worse and I was called about 4:00 a. m., at which time the patient's right face and left body were paralyzed. Cultures taken from the throat and nose were negative. In spite of the use of a pulmotor for several hours and prompt administration of hypodermics, the patient gradually became weaker and weaker and finally died a little more than sixteen days from the time of the removal of his tonsils. No outopsy was obtained.

In this case there are three possible causes of death:— 1, Botulinus poisoning; 2, embolism of the brain; 3, infantile paralysis. I am rather of the opinion that the latter was the cause of the death.

I have since seen two cases of infantile paralysis with exactly the same symptoms exhibited by this patient and both cases resulted in death in the same manner.

In conclusion, I would emphasize the statement made in the first part of this paper:— that deaths following tonsil operations should be reported and that it is important to educate the public to know that an operation for the removal of the tonsils is not lightly undertaken and should be done by some one who is competent.

CASE OF SUBGLOTTIC EDEMA DUE TO ACUTE LYMPHATIC LEUKEMIA.*

Dr. L. Hubert, New York City.

Textbooks of laryngology do not consider leukemia as a causative factor in subglottic edema, and medical treatises in general do not mention subglottic edema as a possible complication of leukemia. In some special articles on leukemia, however, it is hinted that there may be a leukemic infiltration of the larynx, as demonstrated post-mortem.

The case to be reported in a unique one. The first serious and demonstrable lesion appeared in the subglottic region of a man, who was otherwise apparently quite healthy and strong, when first seen by me on March 29, 1922.

The patient was 28 years of age, a Hungarian Hebrew, and leather bag operator.

His chief complaint was that he had difficulty in breathing on exertion.

His family and personal history have no bearing on his condition.

He was always in good health until about the middle of January, 1922. At that time he suffered from severe pains over both clavicles. The attending physician thought that he had "rheumatism." The pain was relieved by electric lamp treatment and lasted about one week. He then worked very hard from early morning until late at night, doing overtime work. About the middle of February he began to suffer from severe shooting pains in the right leg, the right wrist, the left wrist and diffuse pains in the head. These pains, after about four weeks' duration, finally disappeared, but the patient noticed that the glands of the neck were somewhat enlarged and that he had some difficulty in breathing. He consulted many physicians, but none could account for his shortness of breath, as his lungs, heart, and abdominal organs were absolutely negative. Finally he was advised to see a throat specialist.

When the patient came to me (March 29, 1922), the only symptom he complained of was difficulty in breathing on exertion. When quiet he would breathe perfectly well. But when he exerted himself or talked a little, a peculiar grunt to his breathing was noticed. It seemed as if there were some obstruction to the expiratory air.

*Read before the Section of Rhinology and Laryngology, New York Academy of Medicine, October 25, 1922.

This was followed by an inspiratory hissing sound. Then his breathing would be normal again. There was no pain in the larynx, no dysphagia and no hoarseness. There was no elevation of temperature at the time of the first examination.*

Physical examination at that time showed a fairly well nourished man, with practically no anemia and no cyanosis.

Nasal examination was negative.

The tonsils seemed to be diseased. On pressure cheesy masses and pure liquid pus was seen exuding from the tonsillar crypts.

Laryngeal Examination: Everything above the vocal cords was absolutely normal. On phonation the vocal cords moved with normal freedom, they met exactly in the median line and concealed the subglottic edema. On deep inspiration two large, oval, symmetrical, uniformly smooth, dark purplish colored swellings were seen below the vocal cords. They were practically in contact, only a very slight fissure remaining through which the patient was breathing.

Glandular enlargement: Not only the glands of the neck, but also the epitrochlear, the axillary and inguinal glands were enlarged. They were freely movable, discrete, sharply outlined, firm, not tender and not adherent to the skin. Their size varied from one-eighth to one-half of an inch in diameter, the larger ones being located in the region of the neck.

Heart and lungs were negative. Liver and spleen were not enlarged. The chest and abdomen showed a macular eruption, similar to a syphilitic roseola. The Wassermann reaction by three different laboratories, however, was negative.

On April 14, 1922, the patient had a severe attack of suffocation and was admitted to the Manhattan Eye, Ear and Throat Hospital. He was very cyanotic and had constant inspiratory and expiratory dyspnea, which was not relieved by steam inhalations and the ice collar. A tracheotomy was clearly indicated. On April 15 Dr. White and I performed a so-called "tranquil" tracheotomy under local anesthesia (novocain). Before the trachea was opened a few drops of a 5 per cent solution of cocain were injected into it between the upper tracheal rings. There were neither pain, spasm, coughing in performing, nor any reaction following this operation. The cyanosis and dispnea disappeared and the patient felt quite well.

Examination of the blood (April 14) showed the following: White blood cells, 18,000; polymorphs, 10 per cent; small lymphocytes, 8 per cent; large lymphocytes, 82 per cent. The blood was

*The subsequent course of the disease was characterized by an irregular fever (99° to 102° F.).

examined quite frequently at the Manhattan Eye, Ear and Throat Hospital and varied only little from the above.

Urine examination: April 15, 1922. There was a trace of albumen, no sugar, a trace of acetone and diacetic acid, no casts.

On April 20 I removed a gland from the neck for microscopic examination. Dr. Eggston, the pathologist of the hospital, gave the following report:

"The specimen consists of a firm nodular encapsulated lymph node. Sections show marked hyperplasia of the lymphoid tissue showing mitotic nuclei. The endothelial cells of the lymph spaces are hyperplastic. The lymph follicles are obscured. Very few eosinophiles and no giant cells are noted. Diagnosis: Lymphadenopathy. Remark: No definite diagnosis is possible from the sections but an active hyperplastic condition is present."

On May 6 the patient was discharged from the hospital wearing the tracheotomy tube. It was decided to try X-ray treatment on the larynx, for which purpose he was referred to the Lenox Hill Dispensary. He remained well for ten days. Then he complained that he felt as if "something was pulling him down." Examination showed for the first time an enlargement of the spleen. The glands of the neck, however, practically disappeared after the first application of the X-rays to the neck and larynx. On laryngeal examination it was noted that the edema below the vocal cords was diminished. The patient could breathe quite well even when the opening of the tracheotomy tube was closed by the finger.

As the patient became very weak, his feet and ankles swollen, he was admitted on May 24, 1922, to the Lenox Hill Hospital for regular X-ray treatment. The first few treatments seemed to do him a great deal of good. Deep Roentgen ray therapy, applied alternately to the neck (larynx), the mediastinum, the liver and the spleen, seemed to improve his condition to such an extent that it was thought for a time that he might recover. The swelling in the feet and ankles disappeared, the tracheotomy tube was removed and the wound healed completely. He ate well, he slept well, and he felt well. This improvement lasted only a short time. The spleen and the liver were increasing in size, the ankles and feet became swollen again, and there was a leukemic infiltration of the entire skin except in those places where the X-rays had been applied. He became very weak; he could not lie down, but had to sleep in the sitting position. On June 13 he suffered from double vision, due to involvement of the right external rectus muscle. He became almost completely deaf in both ears, which was probably due to leukemic infiltration of the labyrinths.

Blood examination at the Lenox Hill Hospital: The percentage of lymphocytes ranged between 90 per cent to 96 per cent. The white cell count was steadily increasing in amount, from 28,400 (on May 24) to 61,400 (on July 18). On July 22, the blood count was as follows: Hemoglobin, 50 per cent; red blood cells, 2,300,000; polymorphs, 4 per cent; lymphocytes, 96 per cent; white blood cells (no record). The patient died on July 23, 1922. There was no autopsy.

Diagnosis: On account of the comparatively low leukocyte count, the diagnosis of lymphatic leukemia was first doubtful. The subsequent course of the disease, however, was undoubtedly one of lymphatic leukemia. Similar cases with low white cell count have been reported in the literature under the name of aleukemic leukemia.

Acute Hodgkin's disease was also considered, but was excluded on account of the very high lymphocytosis and the negative microscopic findings of the excised gland. Besides the glands in this case were discrete and not matted together.

The subglottic edema was most probably a leukemic infiltration of the subglottic tissues or partook of the nature of lymphomata. It was not inflammatory in character and was not due to renal or heart disease. The fact that it was practically dissolved by the X-rays would make one incline to the above belief.

Etiology. Nothing of importance is known as to the cause of lymphatic leukemia, although the possibility suggests itself that the disease may be due to foci of infection in the head or in the intestinal tract. The fact that this patient had diseased tonsils and no intestinal disturbances and that practically the first lesion appeared in the region of the neck, may incline one to the belief of a possible relationship existing, in this patient at least, between the tonsillar infection and this disease. However, there is no definite evidence available that this is true. Some authorities favor the hypothesis of a close relationship between leukemia and the neoplasms.

Comment. The history of this case shows the importance of making a blood count in every obscure condition. If the diagnosis had been made earlier and X-ray treatment applied before the extreme state of suffocation occurred, the opening of the trachea might have been dispensed with and the patient's life might have been prolonged or possibly saved.

161 East 79th Street.

THE NEW YORK ACADEMY OF MEDICINE

SECTION ON LARYNGOLOGY OF MEDICINE.

January 24, 1923.

Plasma Fibroma of the Larynx. Dr. Francis W. White.

H. S., age 46, was born in Austria and it is a recognized fact that benign growths of the larynx are more common among native born French and Germanic peoples, than among native born Anglo-Saxons. No doubt many of you have been impressed by this fact from observations in the larger continental clinics.

The patient presented himself at the Manhattan Eye, Ear and Throat Hospital in April, 1922 for treatment, and was assigned to Dr. McCullagh's Clinic, where a tentative diagnosis of fibroma of the larynx was made. He had been hoarse 19 years. Fifteen years ago an intranasal operation had been performed, and two years ago a tonsillectomy. He still had two important factors in the production of benign growths of the larynx, namely, nasal obstruction and sinusitis. For the past three weeks (last April) the hoarseness was much worse. No particular improvement occurred under local treatment, and syphilis and tuberculosis having been excluded, a section of the growth was removed by the indirect method, from the pink somewhat oval mass over the base of the right true cord and neighboring portion of the corresponding arytenoid. The microscopic report is as follows:

Sections show a slightly hypertrophied stratified squamous epithelium. Subepithelial tissue shows dense hyaline connective tissue surrounding small and large areas of plasma cells. A few eosinophiles are present. None of the cells show active mitosis.

Diagnosis: Plasmo-fibroma. This is a slow growing recurring non-malignant tumor.

Radium treatment at General Memorial Hospital. Patient complains of more soreness and considerable difficulty in breathing. There was the usual reaction to be seen after the first application of radium. So far he has had 4 treatments, all external, and, as far as we can see there is no improvement.

One point of interest is the fact that no hair has grown on his chin below the rami of the inferior maxillae since the first application of radium. Since the second application of radium his gums have been tender and it is painful to masticate. Complains more now of pain in throat than formerly.

DISCUSSION.

Dr. Forbes said the pathology of the condition was new to him; the use of radium was interesting to all. It would be of still further interest to know the method in which the radium was applied; probably externally as well as within the larynx.

Enlargement of the Thymus Gland Treated by X-Rays. Dr. L. Hubert.

J. T., was referred to the throat clinic of the Lenox Hill Hospital for removal of adenoids on May 20, 1921. At that time he was six years old.

Chief complaint was that he could not breathe well, especially at night. He had been examined in various clinics and by private physicians, all thinking that his trouble was due to his adenoids.

Previous History: The child had practically all diseases of childhood, namely, measles, German measles, whooping cough, chickenpox, pneumonia, and was subject many times to colds and grippe. He also had rickets, was bowlegged, and had to wear a brace for nine months. He had often spells of fainting, when he would stop breathing and appear as if not alive. When he was four years old, his tonsils and adenoids were removed in one of the Board of Health Stations on August 14, 1919. Following the operation he was very ill, vomited for four days, had fever, which at one occasion was as high as 104 F. He passed only small quan-

tities of dark colored urine. Three weeks after the operation he was admitted to Mount Sinai Hospital. There it was noted that he had not passed urine for 26 hours. Examination of the child at that time showed a puffiness of the face. Otherwise the physical examination was recorded practically negative. Examination of the urine showed a trace of albumin and a few white blood cells. The blood chemistry was as follows:

urea nitrogen.....25.2 milligrams per 100 cc blood.
non coagulable nitrogen.....63 milligrams per 100 cc of blood
uric acid.....2.5 milligrams per 100 cc of blood.
creatinine.....1.2 milligrams per 100 cc of blood.

The phenolsulphonephthalein output was 72%.

The diagnosis at Mt. Sinai Hospital was acute nephritis. The child was discharged six weeks after admission.

Present Complaint. On close questioning the mother of the child I found that the trouble was not so much a difficulty in nasal breathing, but that there was a dyspnea, inspiratory in type, coming on especially at night or after exertion and accompanied at times by attacks of suffocation.

Examination showed that the tonsils were well removed. There was practically no adenoid tissue as determined by digital examination. The septum was thickened and deviated. Percussion over the upper part of sternum revealed marked dullness.

Physical examination in general showed certain signs of mal-development, which have been associated with status thymolymphaticus. His upper extremities are too long for his thorax. Teeth: The lateral incisors are small compared with the central incisors. The canine teeth are flat instead of being fanged. There is a hyperextension of the phalanges. He also has falling of the plantar arches, flat feet.

The diagnosis of enlargement of the thymus gland was confirmed by X-ray examination. It showed a quadralateral shadow occupying the upper and midpart of the chest. This shadow can distinctly be separated from the heart shadow. The roentgenologist of the Lenox Hill Hospital believed it to be a thymus gland.

Urine examination at the present time is entirely negative.

Treatment. It consisted in deep X-ray therapy. He had about a dozen such treatments. A subsequent X-ray photograph, shows that the thymus gland is undergoing a state of dissolution. The definite well defined border of the thymus gland in the photograph before treatment is gone. The attacks of suffocation are also gone, but the child still has some difficulty in nasal breathing, which is due to the deviated septum and sinusitis.

Comment. The case shows the importance of taking a careful history and of ascertaining the exact nature of the respiratory difficulty. It also shows that a patient with an enlarged thymus can be operated upon without necessarily a fatal outcome. It is possible that this child was suffering from an acidosis after the operation. It is known that patients of the status thymolymphaticus group are very prone to develop this condition, as they usually have a low carbon dioxide tension in blood plasma. On a basis of acidosis we can explain the symptoms that followed the tonsillectomy, the more so as the laboratory findings did not absolutely point to an acute nephritis. It is also well to remember that Martin H. Fischer in his book on Oedema and Nephritis calls attention to his observations that all the changes in nephritis are due to an acidosis or better expressed, to a diminished alkalinity of the blood in the kidney and general circulation.

Enlargement of the Thysus Gland. Dr. L. Hubert.

A. K., five months old, male, was brought to the Manhattan Eye, Ear and Throat Hospital on January 4th, 1923.

Chief complaint was that the child had difficulty in breathing.

Family History: Mother is 17 years old, father 30 years old, both of good health. This is their first child. It was delivered by caesarean section in the Manhattan Maternity.

Present trouble started three weeks after discharge from the Maternity. The mother noticed that the child had difficulty in breathing, especially at night. When the child cannot breathe or when it cries it gets blue in the face and has attacks of suffocation. These attacks are also brought about by a dry cough, which the child has. The child was examined several times at a pediatric clinic and by private physicians, but nobody seemed to understand the underlying condition.

When the child was brought to the Manhattan Eye, Ear and Throat Hospital, the history the mother gave made us suspect a possible enlargement of the thymus gland. Nose and throat examinations were practically negative. There was some dullness on percussion over the manubrium sterni and a congenital angioma on the scalp.

X-ray examination at the Manhattan Eye, Ear and Throat and Lenox Hill hospitals showed just above the heart shadow an area of density extending on both sides of the vertebral column, which suggests an enlarged thymus.

H. E., 18 months old, male, was brought to the Manhattan Eye, Ear and Throat Hospital on December 23, 1922.

Chief Complaint: Difficulty in breathing.

Family History: Mother had six children, four alive and well. One died one day old and one was born dead due to difficult labor, a version being performed.

Previous History: The child was born in the Harlem Hospital. The mother was in labor only ten minutes. It was a breech delivery. For several days it was difficult to get the child to breathe and he could not take the breast for two weeks. He had to be kept in an incubator, although the pregnancy lasted fully nine months. The child began to sit up when ten months old, but does not walk yet. He cannot say any words, but only mumbles.

Present trouble started when the child was born. Three or four times during the night he wakes up, and has attacks of inspiratory dyspnea.

At a children's clinic the mother was advised to have the child's tonsils and adenoids removed.

Examination at the Manhattan Eye, Ear and Throat Hospital showed that the child had tonsils and adenoids, but their size were not sufficient to explain the attacks of dyspnea.

Furthermore the child shows a hyperextension of the phalanges, and a hyperextension of the knee joints. The latter condition might explain why the child does not walk.

X-ray examination at the Manhattan Eye, Ear and Throat and Lenox Hill hospitals showed above the heart shadow an area of density extending on both sides of the vertebral column, which suggests the presence of a thymus gland.

Substernal Thyroid With Interesting Laryngeal Complications. Dr. L. Hubert.

D. G., 50 years old, born in Poland, came to the Manhattan Eye, Ear and Throat Hospital on January 7, 1922.

Her chief complaint was difficulty in breathing and choking attacks.

Family history has no bearing on her trouble with the exception that her grandmother had a goiter.

Previous History: She had pneumonia three times. Otherwise she was in good health up to eight years ago when her menapause begun. Since that time she became very nervous, had palpitation of the heart on exertion, felt very weak and lost considerable weight. A physician from the Board of Health sent her to the Bedford Hill Sanitorium, where she remained for six months. She had no cough, but lost more weight, altogether about 35 pounds. From her usual weight of 125 pounds she was reduced to 92 pounds. Dr. Alfred Meyer, who examined her at the Sanitorium, was convinced that she had no tuberculosis and advised her to go home.

Present History: The difficulty in breathing and choking attacks started about two years ago. A laryngologist thought that her trouble

was due to nasal obstruction and he performed a submucous resection of the nasal septum. As her symptoms became worse she was advised to have her tonsils removed. She then came to the Manhattan Eye, Ear and Throat Hospital. Besides the dyspnea and choking attacks, she also complained of frequent colds, profuse nasal discharge and slight hoarseness for the last two months. She had similar attacks of hoarseness in the last few years.

Examination at the Manhattan Eye, Ear and Throat Hospital: Patient is fairly well nourished, weighs 150 pounds. There is a fine tremor of the tongue and outstretched fingers and a pulse rate of 120. Dullness on percussion over upper part of sternum.

Laryngeal Examination: The left cord does not move at all, and is shortened. The left arytenoid is fixed and tipped slightly forward There seems to be a complete left recurrent laryngeal paralysis. The right cord does not abduct, but in phonation crosses the median line and approaches the left cord. This compensation explains her fairly good voice.

X-ray Examination, January 10, 1922. The trachea seems to be in the median line, although there is a possible slight deviation to the left. There seems to be a mass in the superior mediastinum which may be a thymus or a substernal thyroid. There is also some scoliosis from the second to the tenth dorsal vertebrae.

Wassermann reaction negative.

Fluoroscopic examination on March 1, 1922 reveals no further involvement. Dr. Law thinks that the substernal mass is a thyroid.

Treatment. The patient had four deep Roentgen ray treatments. This treatment did not seem to do her any good subjectively. After each treatment she had to be in bed for three to four days on account of the extreme weakness. This weakness seemed to be increasing after each treatment. Her pulse rate and nervousness were not diminshed. Objectively the X-rays seemed to have done some good, as shown by another X-ray picture, taken on November 14, 1922. The shadow is considerably diminished in size. The laryngeal picture, however, has not changed. She had no X-ray treatments for about six months, as she is unwilling to have them. Since November 11, 1922, she is getting quinin hydrobromide, gr. V. t. i. d. She feels somewhat better, is not so nervous and her pulse rate is reduced to 98.

DISCUSSION.

DR. CARTER said that Dr. Hubert had brought up a very interesting subject, that of status lymphaticus and enlargement of the thymus gland. Personally he felt that he was not qualified to say whether or not the X-ray is capable of reducing the size of an hypertrophied gland; Dr. Law was more competent to discuss that matter, but it did not seem to him that Dr. Hubert correctly interpreted the diminution in the size of the gland in his case, it might be due to the normal atrophy which occurs at this age. The history of the thymus is that it continues to enlarge up to the age of two years and then begins to atrophy and practically disappears at puberty, though it may remain, very much diminished in size, throughout life. If the X-ray proves to be effective in diagnosing an enlarged thymus it will be of great value, especially where the adenoid and tonsil operation is being considered, for unsuspected status lymphaticus is a serious menace to life in this operation. Dr. Carter said that three deaths had occurred from status lymphaticus in his hospital practice; one died from tracheostenosis and the other two from toxemia due to the enlarged thymus. The child that died from tracheostenosis was five weeks old. Tracheotomy was performed and a long tube inserted, but the child died three days later from broncho-pneumonia. It is useless in these cases to put in a short tube, the tube should be long enough to reach to the bifurcation of the trachea.

The two cases that died with toxic symptoms were operated upon for adenoids and tonsils, and each died about three hours after the operation. In both instances autopsies were performed, and there was no

question about its being status lymphaticus. In one case the thymus weighed 21½ grams. The question would arise whether or not it was possible to diagnose any of these cases beforehand. In the two toxic cases there were no symptoms to indicate that the patients were suffering from status lymphaticus. In a large clinic such as Dr. Carter's, where from fifteen to twenty-five tonsil and adenoid operations are frequently performed in a single afternoon, it would be quite an undertaking to exclude thymic enlargement. Certainly no patient known to have an enlarged thymus should be operated upon under a general anaesthetic.

DR. LAW said that the X-ray diagnosis in these cases was rather difficult unless there was considerable increase in the size of the shadow over the heart. There is normally a broader shadow here in young children and infants, but just where the normal size begins to become abnormal is sometimes difficult to state. A marked increase in the size, however, is suspicious. Stereoscopic plates are necessary, and these are difficult to make in the case of an infant.

The X-ray has a marked effect in shrinking the size of the thymus, and any case which shows a considerable increase in the thymus shadow is surely deserving of the benefit of such treatment.

DR. MCCULLAGH said there could be no doubt that in certain cases of enlargement of the thymus the patient may go through an operation satisfactorily; not every case of status lymphaticus or thymus enlargement terminated fatally on the operating table. Dr. Hubert had cited one case, and Dr. McCullagh recalled another case in private practice where there was no question about the status lymphaticus. The child was brought to him for treatment of an acute otitis, for which a paracentesis was done, and the adenoids were removed with no untoward effect. Later, the child was brought to the office and had a typical thymic attack which was very alarming. Light is needed upon the limits in the size of the thymus which may be considered normal. In the older cases subinvolution, may be the explanation rather than enlargement. There is no doubt the cases that have had X-ray treatment have shown improvement.

DR. J. J. WALSH said that there was no question about this enlargement of the thymus gland causing trouble, but he was inclined to think that the trouble was most likely caused by occlusion. When he was in charge at Fordham Hospital a child was brought into the service with a history of not being able to breathe with the head in the normal position. It was quite puzzling for a while to determine what the trouble was. There was a dull percussion region over the thymus and the X-ray showed a shadow. The child was operated upon and the thymus was removed and the child was then able to assume the normal position and breathe normally, and remained that way for a year. Finally the parents removed with the child to Texas and there it died of pneumonia. It seems probable that the thymus gives trouble from the fact that its enlargement causes obstruction of the respiratory passage.

DR. FORBES said that the thought had occurred to him that the peculiar condition may be existing at the time and yet not always present the symptoms; it may exist as an hypo or a hyper secretion. It was his impression that most of the status lymphaticus deaths occurred rather suddenly after operation, and were not associated with dyspnea, and they were apt to be associated with enlargement of the thymus.

DR. HUBERT said that from among all the cases who came to the clinic to have their tonsils and adenoids removed, only about 10 were referred to Dr. Law for X-ray examination of the thymus gland. He thought that when children were brought to the hospital with the history not only of difficult breathing, but also of attacks of suffocation, more care should be exercised with them. They do not necessarily die when operated upon, but accidents are liable to happen. Cases belonging to the status lymphaticus group do not necessarily have an enlarged thymus gland. Some of these cases were X-rayed by Dr. Law and he reported only a faint suspicion of an enlarged thymus. These children looked healthy

and fat, yet they were very weak. They complained of nasal obstruction. Such cases should not be operated upon, but treated with X-rays.

DR. KING told of a similar case which had been referred to him one year previously by a surgeon who had removed the right lobe of the thyroid, after which the patient became very hoarse. The patient stated that she had been hoarse for a year, and examination revealed a total paralysis of the left vocal cord. That case emphasized the point that all of these thyroid cases should be examined by a laryngologist before thyroidectomy is performed. At the Cleveland Clinic, Crile states that about 10 per cent of goitre cases have laryngeal involvement. It may be due to toxemia or it may be due to pressure.

In cases with total paralysis of one or both vocal cords the prognosis is not good. We know of no treatment which will restore the functions of the cords. Stimulation by tonics and electricity have been recommended, but the improvement in function is doubtful. Chevalier Jackson has recommended excision of the cords in paralysis of both vocal cords.

DR. FORBES said that the question of paralysis in thyroid cases interested him greatly, for within a few years the subject had come up at the Post-Graduate Hospital with reference to cases treated by the X-ray. It so happened that several of the patients had paralysis and it was a matter of discussion whether or not it was due to actual contraction of the connective tissue following X-ray treatment; it is now the practice there, to thoroughly examine all the patients before they are subjected to X-rays, and an effort is being made to work out a method of procedure for these cases.

An Interesting Case of Laryngeal Stenosis. Dr. E. M. Josephson.

(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

DISCUSSION.

DR. EMIL MAYER said that he had not been able to follow the case report closely because of difficulty of hearing the speaker, but his understanding was that the condition was an inflammatory one and that it was said to be non-specific because it had not yielded to mixed treatment. Nowadays this form of treatment is not sufficient to effect results or to rely upon.

The Wassermann was said to be 4 plus. The swelling had not broken down. This last in itself is not absolutely diagnostic.

The description fits in however in point of similarity to a peri-chondritis of the larynx, such as occasionally followed typhoid fever, of which Dr. Mayer had reported a case that was successfully treated. If the facts were as the speaker understood it could easily be classed as a gumma that had not reached the stage of breaking down.

DR. JOSEPHSON regretted that there were no microscopes at hand for demonstration of the slides sections of the tumor, micro-photographs of which he had not been able to prepare. The tumor presented, on section, an appearance which might be compared with that of a chronic nephritis, a mass of scar tissue in which was interspersed the remains of tubules.

With regard to the ruling out of lues from the diagnosis, in the presence of a Wassermann, the following excellent reasons are to be held in mind: First, anti-luetic treatment including salvarsan treatment, mention of which was, by oversight, omitted in the paper, had been of no avail; and that is contrary to what one would expect of a gumma. Secondly, the acute inflammatory onset is not a usual thing in tertiary lues. Third, tertiary luetic lesions of the larynx give a rapidly changing picture, and usually caseate long before attaining the size of the tumor in this case, resulting in ulceration of the mucous membrane and scar formation; in this case, the lesion had remained stationary and unchanged for several months. Fourth, there was absolutely no sign of other tertiary luetic lesions in the respiratory tract, such as one would expect to find in tertiary lues involving that tract. Finally, the microscopic picture showed no sign of gumma formation or of caseation.

Surgical Emphysema Following Tonsillectomy. Dr. Simon Stein.

(To appear in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. KING told of the case of an adult upon whom, about a year ago, he had done a tonsillectomy under general anesthesia followed soon afterward by emphysema of the face and neck. Dr. Bennett gave the anesthesia and the operation was carried out quickly with very little trauma and almost no bleeding. My attention was called to the swelling of the lady's face a few hours later by her husband. A distinct crepitus was felt in the tissues, and she was told it was an emphysema but I had no idea how to account for it. I had never seen nor heard of it following a tonsillectomy or an anesthetic. It passed away in the course of the next 48 hours. In view of this experience I was much interested in Dr. Stein's report.

DR. LEWALD said that Dr. Stein had mentioned exploratory puncture of the chest as a possible cause of subcutaneous emphysema, and he then referred to two cases of that sort at St. Luke's Hospital where a supposed emphysema that was not submitted to X-ray study but was punctured for diagnosis resulted in a very extensive emphysema which very nearly ended fatally; the child died subsequently of pneumonia. In that case, the emphysema did not go down readily. In another case there was a rapid subsidence of the emphysema and the unresolved pneumonia also cleared up.

(To be Continued.)

Program of the forty-fifth Annual Congress of the American Laryngological Association held at the Hotel Ambassador May 16-18, 1923.

President's Address. Dr. Emil Mayer, New York City.

1. "Acute Laryngeal Edema." Dr. Clement F. Theisen.
2. "A Surgical Method of Relieving Abductor Paralysis Without Destroying the Voice." Dr. John E. Mackenty.
3. "Fluoroscopic and X-Ray Studies of the Diaphragm and Esophagus." Dr. Harris P. Mosher.
4. "The Relation of Sinusitis to Arthritis Deformans—with Report of Cases." Dr. E. Ross Faulkner.

SYMPOSIUM

5. "Chronic Tonsil Pathology and Its Relation to Systematic Infection." Dr. George B. Wood.
6. "Chronic Infections of the Upper Respiratory Tract and Their Relation to General Disease." Dr. Francis P. Emerson.
7. "The Effect of Dental Infection on the Rest of the Body." Dr. Kurt H. Thoma, Boston, Mass.
8. "The Nature of the Influence of Focal Infection and the Means Necessary to Meet It." Dr. Ralph Pemberton, Philadelphia, Pa.
9. (a) "Anatomy of the Sphenoidal Fissure." (b) "Glossodynia with Lingual Tonsillitis as its Etiology and Its Control Through the Nasal Ganglion. Report of a case." Dr. Greenfield Sluder.
10. "Glossal Pharyngeal Neuralgia." Dr. H. I. Lillie.
11. "Notes on Fourteen Cases of Intrinsic Cancer of the Larynx with Lantern Demonstration of the Pathological Anatomy." Dr. J. S. Fraser, Edinburgh, Scotland.
12. "Radical Operation for Extrinsic Cancer of the Larynx." Dr. Vilray P. Blair.
13. "The Reaction of the Paratonsillar Tissues to Tonsillectomy." Dr. George Fetterolf, Dr. Herbert Fox, Philadelphia, Pa.
14. "Further Experience with the Use of Tissue Juice in Tonsillectomy." Dr. Joseph B. Greene.

15. "Stenosis of Larynx and Trachea. Treatment." Illustrated by Moving Pictures. Presentation of Patient. Dr. Francis W. White.

16. "Brain Abscess of Paranasal Sinus Origin. Two Cases." Dr. Gordon Berry.

17. "Fibroma of the Naso-Pharynx. A Clinical and Pathological Study." Dr. Perry G. Goldsmith.

18. "A Case of Epithelioma of the Esophagus with Some Unusual Features. Specimen." Dr. Samuel McCullagh.

Program of the twenty-ninth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., at The Ambassador, Atlantic City, N. J., May 10, 11 and 12, 1923.

1. "Operative Obliteration of Both Lateral Sinuses with Recovery." Dr. H. G. Tobey, Boston Mass.
2. "Alcohol Injections As a Possible Arjunct to Tonsillectomy Under Local Anaesthesia." Dr. Robert Sonnenschein, Chicago, Ill.
3. "Some of the Aspects of Aphonia, Psychic and Otherwise." Dr. J. A. Babbitt, Philadelphia, Pa.
4. "Laryngeal Epilepsy." Dr. Beaman Douglas, New York City.
5. "Nasopharyngoscopy Under Negative Pressure." Dr. R. P. Scholz, St. Louis, Mo. Candidate's Thesis.
6. "A Study of the Effect of Roentgen Ray Therapy Upon Impaired Hearing." Dr. D. C. Jarvis, Barre, Vt.
7. "Submucous Resection of the Nasal Septum in Children." Dr. F. W. White, New York City.
8. "Some Observations on Throat Mycoses." Dr. G. E. Davis, New York City.
9. "Routine Procedures and End Results in 3000 Tonsillectomes." Dr. S. H. Large, Cleveland, Ohio.
10. "Surgery of the Frontal Sinus." Dr. C. T. Porter, Boston, Mass.
11. "The Otological Aspects of Recklinghausen's Disease." Dr. M. A. Goldstein, St. Louis, Mo.
12. "An Anatomical and X-Ray Study of the Optic Canal in Optic Neuritis." Dr. L. E. White, Boston, Mass.
13. "Intra Septal Implantation in Atrophic Rhinitis." Dr. H. L. Pollock, Chicago, Ill. Candidate's Thesis.
14. "The Diagnosis, Differential Diagnosis and Prognosis of Laryngeal Tuberculosis." Dr. F. R. Spencer, Boulder, Colo.
15. "A New Method of Closing the Eustachian Tube in the Radical Mastoid Operation." Dr. N. H. Pierce, Chicago, Ill.
16. "A Lantern Demonstration of the Normal Histology of the Internal Ear." Dr. G. E. Shambaugh, Chicago, Ill.
17. "A Lantern Demonstration on the Subject of Fractures of the Temporal Bone, War Injuries, Labyrinthitis of the Middle Ear Origin, Tuberculosis of the Ear, Otosclerosis, Neuritis and Tumors of the Eighth Nerve." Dr. J. S. Fraser, Edinburgh, Scotland. (By Invitation)
18. "A Study of the Mechanical and Chemical Properties of the Sand Spur from the Standpoint of the Endoscopist and Some Observations on Its Clinical Manifestations in the Larynx." Dr. H. M. Taylor, Jacksonville, Fla. Candidate's Thesis.
19. "Observations on 1135 Foreign Bodies in the Air and Food Passages." Dr. Chevalier Jackson, Philadelphia, Pa.
20. "Dental Complications of Radical Operation on the Maxillary Antrum." Dr. Robert H. Ivy, Philadelphia, Pa. (By Invitation)
21. "Further Studies of Fatalities Following Operations on the Nose and Throat." Dr. H. W. Loeb, St. Louis, Mo.
22. "An Improved Technique in the Operation for Cleft Palate." Dr. J. E. MacKenty, New York City.